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Table of Contents

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ORIGINAL ARTICLES— PAGE.
The Beattie-Smith Lectures: II.—"The Treatment of Insanity by the General Practitioner", by JOHN K. ADEY, O.B.E., M.B., B.S. 145
"A Bacillary Disease of the Blue-Tongued Lizard (*Tiliqua scincoides*)", by RUPERT A. WILLIS, M.D. 149
"Injuries and Diseases in Australia Attributable to Animals (Other than Insects)", Series IV, by J. BURTON CLELAND, M.D. 157

REPORTS OF CASES—
"Some Notes on a Case of Arsenical Polyneuritis", by GREY EWAN, B.Sc., M.B., Ch.M., Dipl.Psych. 166

REVIEWS—
A Guide for Diabetics 167
The Literature of the Vertebrates 167
Sex Life 168
Histological Methods 168
Another Book on General Practice 168

NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—
Another Book on Russia 168

LEADING ARTICLES—
Obstetrics 169

CURRENT COMMENT—
Betel-Nut Cancer 170
The Latest Specialty 171

ABSTRACTS FROM CURRENT MEDICAL LITERATURE— PAGE.
Pædiatrics 172
Orthopædic Surgery 173

BRITISH MEDICAL ASSOCIATION NEWS—
Scientific 174
Nominations and Elections 176

PUBLIC HEALTH—
Queensland 176

CORRESPONDENCE—
Spinal Analgesia 177

OBITUARY—
Mervyn Athol Stewart 178

MEDICAL PRIZES—
The Alvarenga Prize 179

POST-GRADUATE WORK—
Lectures in Adelaide 180

BOOKS RECEIVED 180

DIARY FOR THE MONTH 180

MEDICAL APPOINTMENTS VACANT, ETC. 180

MEDICAL APPOINTMENTS: IMPORTANT NOTICE 180

EDITORIAL NOTICES 180

The Beattie-Smith Lectures.[1]

(UNIVERSITY OF MELBOURNE.)

THE TREATMENT OF INSANITY BY THE GENERAL PRACTITIONER.

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LECTURE II.

Obsessions.

WE now come to the form of mental disorder which is not insanity and is rarely seen in asylums. I refer to obsessions. The distinction between the obsessional and the insane is that the former admits that the obsessions are untrue and absurd, but the insane never admit that there can be anything untrue in their beliefs. We all have experienced obsessional ideas, for instance a particular tune will keep running in one's head and for some hours it is impossible to shake it off. Mark Twain's story of "Punch, Brothers, Punch" is a story of a typical obsession. If you imagine an imperative idea of this sort possessing the mental faculties for hour after hour and day after day, you can possibly imagine what mental distress this may cause. Obsessions may occur at any age and affected persons very rarely become insane; reassurance on this point frequently does them a great deal of good.

This type of condition is often extraordinarily intractable and the only type of treatment which does any good is psychoanalysis.

Psychoanalysis.

Psychoanalysis which was started about twenty-five years ago by Freud, of Vienna, has been the subject of much comment and acrimonious discussion in both the lay and the medical Press. It is im-

[1]Delivered on October 19 and 22, 1931.

possible to give the whole theory of psychoanalysis, but the main points about it are that the whole past mental history of the patient is examined by a study of his past experiences, his dreams, his thoughts and phantasies. It is then found out that he has experienced some deleterious sexual positions or peculiarities, what are called by the school "sexual fixations", often of an incestuous nature, and that these experiences always date back to the first seven years of childhood. This has aroused a storm of objection on the part of many of the British psychiatrists, who stated that it was a purely mythical invention of a dirty-minded Austrian. The psychoanalysts reply: "Try it for yourself and you will get results." This in my opinion is true, for I have tried it and I have sometimes got results. The opposition at once reply that the results are obtained, not on account of the psychoanalysis, but on account of long attention to the patient.

I think that, from the scientific point of view, psychoanalysis is quite unwarranted. It builds up theory after theory on most insecure foundations, but at the same time it must be admitted that from the practical point of view it is the only attempt made to solve the difficulties of the obsessional patient. Before Freud started his work on psychoanalysis we stood helplessly before these persons, talking platitudes to them and finally we had to admit that nothing could be done for them. We can now by means of this system obtain an insight into the patient's condition, can understand it, can show the patient that we understand it, and can do a great deal towards improving, if not curing it.

Unfortunately there are only a restricted number of patients suitable for this treatment. The patient must be anxious to get well and this is not true of all patients who consult a doctor. Some of them have taken a flight into disease from an unbearable situation, and would rather have the disease than the consequences attending its cure. This also applies to all persons with genuine insanity, as they do not recognize themselves to be ill. The patient must also possess a certain amount of intelligence or he will not be able to cooperate with the psychoanalyst, and the patient must not be too old, for the mental habits of a lifetime cannot be altered in old age.

Melancholia.

Melancholia is the form of insanity which can be treated in a private house. In the first place the patient often recognizes that he is ill, and is willing to submit to treatment. It chiefly occurs in women about the time of the climacteric, but may occur in anyone at any age. Melancholia in young people, say from the age of twenty to thirty-five, is most satisfactory to treat and usually in a few months a good recovery occurs. Unfortunately when melancholia occurs in later life, the patient sometimes remains permanently enfeebled.

The great principle in the treatment of these patients is to increase their weight by means of rest, food and sleep. The patient should be confined to bed, preferably in the open air, and should be kept free from all mental and physical exertion. The well-meaning visits of anxious and fussy relatives must be forbidden. They insist on telling the patient to "buck up" and to rouse himself, and to take an interest in things; they might just as well tell a man with a broken leg to go in for athletic exercises. The main idea of the relatives is to take the patient to theatres or to places of so-called amusement. If you are feeling in a miserable state of mind, are worrying over something and someone insists on you going to a comic opera with him, I am sure you will agree that you will not enjoy the play, and will only have the mental torture of trying to concentrate on the scene added to your other troubles. In the same way a melancholic does not need amusement added to his other worries.

Visitors must be carefully selected and instructions should be given in writing to the nurse as to who is to be admitted. Sometimes the advice of the nurse is very valuable, as she can see who worries the patient, and which friends have a soothing, rather than a disturbing influence on him.

In the same way a sea voyage or a change to a strange place is quite contraindicated for an incipient melancholic. The worry of the travel, the excitement of seeing new places all increase his depression and it is not until he is well on the road to convalescence that these will do him any good.

Bed treatment therefore, preferably under the care of a tactful and experienced nurse, is the first essential. Food should be plentiful and of all kinds—three meals a day including meat and plenty of fat, and extra milk and eggs between meals. Weigh the patient once a week and if he be not increasing in weight, he is not improving. Sleeping draughts should be given, and in my opinion we are too frightened of the possible after effects of sleeping draughts. They are very necessary at the commencement of the disease and their use can be discontinued gradually as the patient's condition improves. Paraldehyde is the best for routine use; sulphonal, trional or veronal can be given in emergency to start the habit of sleep; bromides, I think, increase the depression. I should warn the doctor that frequently the patient appears to sleep because he lies quietly with his head beneath the blankets, whilst he is really lying awake. The nurse will report that the patient had several hours sleep when he has not slept. She must be warned that it is not easy to determine whether the patient is really asleep.

The question of drug treatment, apart from sleeping draughts, must now be considered. The English school under Clouston had a great objection to opium, saying that it spoiled the patient's appetite without improving his depression; the German school gives opium in all cases of melancholia, and holds that it decreases mental pain. In my opinion opium suits some patients but not all. I usually give 0·18 mil (twenty minims) of opium three times a day and gradually increase it to 0·24 mil (thirty minims) three times a day if it improves the patient's condition and he puts on weight. Tincture of opium or *liquor opii sedativus* is the most suitable. If his condition does not improve, I do not persevere with it for longer than two weeks. If opium is not given, tonics such as iron and arsenic can be substituted, but more for the moral than the actual

effect. Cod liver oil and malt are undoubtedly good, but come more under the heading of food than of medicine.

The bowels must of course be kept well open and it is wonderful to see what calomel and sulphate of magnesia will sometimes do in the way of improving the patient's mental condition. Many cases of melancholia, especially in women, are due to intestinal toxæmia, and intestinal antiseptics after a preliminary purgation are all that is required.

The great danger in treating these patients in a private house is the possibility of suicide. If the patient is actively suicidal, the only way to prevent suicide is never to leave him by himself for a moment. This, of course, entails three nurses; two are not enough, as if there be only one for day and one for night duty, the patient must be left at times. It is difficult to tell whether the patient is really suicidal, and the danger is not past because his mental condition appears to be improving. Sometimes the patient has insufficient will-power to commit suicide in the acute stage, but as his condition improves he is able to scheme methods of destroying himself that would be quite impossible in the earlier stages. Each case must be considered on its merits, but the physician must remember that every melancholic is a potential suicide, and all ordinary precautions like removing razors, seeing that medicines are not kept in his bedroom, and that all antiseptics are kept out of the way, should be taken. The possibility of suicide is another reason for not sending the patient for a sea voyage.

Delusional and Hallucinatory Insanity.

We have now dealt with many forms of insanity that may occur between the ages of fifteen and fifty, but there is one important form that may occur at any age from twenty onwards, but most usually after the age of thirty. I refer to delusional insanity, so dear to the hearts of the legal profession. Apparently to the legal mind, if a man has a delusion he is insane, if not, he is sane. This of course, from a medical point of view is quite incorrect, as many melancholics are very depressed but by no means delusional.

In true delusional insanity the delusions must be at the base of the trouble. If the patient is very excited or profoundly depressed, he is almost certainly not suffering from delusional insanity. One important point to distinguish the melancholic from the sufferer from delusional insanity is that the melancholic blames himself for his misfortunes, the delusional patient blames everyone else.

Delusions and hallucinations are due to pathological processes and consequently it is quite useless to argue with the patient and to try to convince him that his beliefs are absurd. The treatment of these patients is useless. When a physician is called to see a delusional patient, his actions must be governed by the actual facts. The late Dr. Charles Mercier defined insanity as disorder of conduct, and this definition has the supreme advantage of being a very practical one. If the patient indulges only in harmless eccentricities, there is no need to certify him as insane. The circumstances under which the patient lives have a considerable bearing on his treatment. If his family is sincerely desirous of keeping him out of an asylum it is surprising what a lot can be done to manage this type of patient. The patient's conduct with regard to the general public must be considered as well as his conduct at home. It is impossible to have a person with delusional insanity in a crowded suburb if he insists in accusing his neighbours over the backyard fence, whilst a similar patient in the country may be managed with ease.

In other words, a man should not be certified because he is suffering from delusional insanity, but because he is unable to live in peace, love and charity with his relatives or his neighbours.

Physical Diseases.

I now wish to deal with cases in which the insanity is in relation to physical diseases. These can be summed up under two headings: (i) those cases in which the disease only accentuated a latent or approaching insanity, and (ii) those cases in which the disease was the actual cause of the insanity and without which the patient would not have become insane. Those of the first type have already been dealt with under the various types of insanity. Thus an attack of influenza may appear to cause delusional insanity, because the added stress of influenza has been sufficient to make the patient lose control of himself and so to show delusions he had previously concealed.

Those of the second type in which the physical disease is the actual cause of insanity, will now be considered.

I here wish to call attention to the work that can be done by a general practitioner in relation to the mental symptoms in general diseases. We all know the apathy of the patient with enteric fever, or the irritability of the man with cardiac failure, but there are probably many diseases with characteristic mental accompaniments which are not generally recognized as part of the disease. What is the mental reaction of patients with rheumatoid arthritis? Do patients in different positions in life react differently to physical disease? How does the mental condition of a girl of twenty in a wealthy household compare with that of a girl in a factory, when they are both suffering from the same complaint? All these are points that can be studied by a general practitioner with a practice containing patients in a wealthy suburb, and also those in a general hospital.

I know that the specialist is accused of seeing in every patient examples of diseases of his own particular specialty, and the alienist must frequently plead guilty to this charge. But everyone who has dealt with mental patients has seen persons whose condition has been due to mental symptoms only, and who has been operated on time and again for various complaints, each operation serving to fixate delusions. Sometimes the physician reasons thus: The patient complains of pains in the stomach, therefore he probably has a pathological condition in his stomach; I shall treat this condition and if the symptoms arise from a purely mental condition it

will not do him any harm. Unfortunately the greatest harm is done by strengthening the patient's delusion and fixing his attention on that particular part of his body.

To return to insanity caused by physical disease. Any acute bacterial infection as pyæmia, septicæmia, pneumonia, enteric fever or malaria may cause an attack of confusional insanity. In these cases the original cause may be very difficult to diagnose, as the patient is often wildly excited and it is difficult to take even the temperature until the patient is under the influence of hyoscine. To make things still more difficult insane patients do not complain of pain, in fact do not seem to suffer it, and it is not impossible to have a patient with acute pneumonia running all over the place. This type of condition can be compared with the delirious conditions frequently seen in children who, when they have a slight rise in temperature, talk incoherently and pick at the bedclothes. As the acute condition subsides the symptoms of insanity pass away, and the treatment consists in treating the original condition.

Diabetes, albuminuria and toxic poisons such as lead and belladonna may cause hallucinatory symptoms. These patients usually improve as the condition is treated.

Influenza often causes extreme depression and this depression often lasts for a long time, and I have been struck by the fact that patients' friends frequently date the onset of an attack of melancholia from influenza. I lay considerable stress on this as I am convinced that many an attack of melancholia could be prevented, particularly in women about the age of forty-five, if the post influenzal depression was recognized as serious and treated accordingly. If allowed to run on, it may progress to a condition of permanent enfeeblement. As a general rule it is very amenable to the ordinary treatment of melancholia I have already mentioned.

Insanity due to cerebral anæmia from heart disease, usually aortic regurgitation or stenosis, is not at all infrequent. The patient becomes very irritable and restless and may develop delusions of persecution. These patients recover if the heart lesion can be successfully treated. Such patients may cause trouble in the law courts by altering their wills at the wrong times for some of their relatives.

Insanity of Isolation.

I shall now mention a form of insanity which is not found in text books, because it is probably never seen in England. It occurs in men who live in the country far from their nearest neighbours, who look after themselves, who have insufficient nourishment and a lack of companionship. One might term it insanity due to isolation. The patient is confused, depressed and frequently suicidal. These patients make an excellent recovery under general treatment, good food, rest, general attention and some tonics in the shape of compound syrup of hypophosphites. They can be well treated by their friends in private houses, provided they are away from their previous surroundings, and they must not return to their original work in the same conditions.

Senile Insanity.

We now come to consider the insanities of old age, and of all forms of insanity I think these are the forms for which the treatment of the general practitioner can be of most value. It is very sad to see a fine old man who has brought up his family in faith and fear, especially fear, have to be sent to an asylum in his old age because he nearly sets fire to the house trying to light matches in bed, and because he loses himself as soon as he wanders a hundred yards from the front door. Recovery of course is impossible, but try to keep him out of an asylum.

Unaccustomed surroundings appear to be particularly unsuitable to old age and these persons are much happier in their own homes. The great difficulty is to manage them at night. During the day a daughter is well able to look after them, but it is beyond her powers to manage both by day and by night. Of course if the friends are wealthy enough to pay for two nurses, there is no difficulty; but we must endeavour to do our best for those who are not favoured with this world's goods.

All senile dements have a tendency to go to sleep after the midday meal. Advantage should be taken of this to give them their midday meal in the evening as our friends from Ireland would say. The heaviest meal should be given at night and the patient should be given some gruel or cocoa or whatever he fancies just before going to bed. If a senile person has been accustomed to alcohol all his life, he must have it in his old age; if he has not been accustomed to it, a hot whisky toddy or a glass of stout just before bed works wonders. All these patients have a tendency to turn night into day, and to wander about at 2 a.m., and some hot food ready to be given them when they wake in the middle of the night will often cause them to sleep quietly till morning. The aged need food frequently and in small quantities. Paraldehyde in doses of 8·0 mils (two fluid drachms) given when they go to bed or when they wake up at night will often assist, but is useless without food.

If these patients can be managed at night their treatment during the daytime is comparatively easy. The old man always wishes to go to sleep after the midday meal and it is sometimes advocated to keep them awake during the day so that they will sleep better at night. Others say that if he does not sleep at night he will not sleep at all. I am of opinion that each case should be treated on its merits, and that wakefulness during the day should be obtained by making the midday meal as light as possible, so that the stomach may not become overloaded.

Any troubles in the shape of high blood pressure or nephritis must of course be dealt with, but if the blood pressure be reduced too much, mental confusion may be caused by cerebral anæmia.

Care should be taken that the patient does not go out alone and lose himself; owing to his loss of memory for recent events he is very likely to be unable to find his way home. This is particularly likely to occur if he has recently moved into a new district, and Anatole France, in "*Le Crime de Sylvestre Bonnard*" gives a delightful example of an old man losing himself in front of his own hotel.

Two other difficulties in dealing with senile patients are suspicion and melancholia. Some old men become inordinately suspicious of their friends and relatives. They suspect them of trying to obtain their money or of trying to deprive them of their homes. In these cases it is advisable, if possible, for the suspected relative or friend to go away for a prolonged period, as "out of sight, out of mind" applies particularly to the senile. On the relative's return the suspicions have sometimes vanished. To try to argue with an old man about his relatives only makes him believe that you are in league with the suspected parties.

In some cases it is necessary to change the nurse frequently. Some patients are at first pleased with the nurse, and afterwards take a quite unreasoning dislike to her. It is no use persisting in such a case, it is better to let the nurse go, no matter how little she has deserved it, and procure another one; for once a senile patient has taken a dislike to his nurse, he will be difficult to manage.

Senile melancholia must be treated in the same way as ordinary melancholia, special precautions being taken against suicide.

Testamentary Capacity.

We have now taken a rapid survey of the perils that beset a man's mental health, but after he has been laid quietly to rest we may have further trouble with him, as his relatives, aided by the legal profession, start fighting over his will. This is much more likely to occur in the case of insanity in old age than in any other.

In order to examine a man to see whether his mental condition is such that he is able to make a valid will, insist on seeing the patient alone. Sometimes a designing nurse or relative has such an influence on an old man that he is quite unable to speak his mind in her presence. Ascertain when the would-be testator is at his best. Some old men are clearer in the morning and some in the evening. If you are not personally acquainted with him, this can usually be found out from his family doctor. Insist on seeing him at the time his mental faculties are clearest.

Ask him how much property he has and how it is distributed, that is, whether it is in land, houses, stocks and shares *et cetera*. He may not be able to give the exact amounts, but if a man who is worth only a few thousands, states that he is a millionaire, he obviously cannot make a will.

Ask him the names of his near relatives and how he wishes the property to be disposed. If he wishes to cut any of them out of his will ask him his reasons, with a view of ascertaining whether any insane delusion is influencing him. He may attempt to conceal the delusion, but in this case his reasons will be absurd, or at least inadequate. If he has previously made a will, ask him why he wishes to alter it. Get him to repeat his statements in other words.

Then see him again in about a week's time and ask him to go over all the particulars about his will, and if his testamentary ideas remain unchanged, you will be in a good position to swear in the witness box that the will should be upheld by the Court.

If the patient is so ill when you first examine him that he may not live a week, allow as long an interval as you think safe to elapse between the first and the second examinations.

Never suggest to the man how his property should be left, nor how the will should be worded, and do not be a witness to the will yourself. Your witnessing the testator's signature will not invalidate the will, but if you have examined him in a professional capacity, it is inadvisable for you to complicate your position by taking a legal part in the business.

Never succumb to the desires of the anxious relatives for you to say that he can make a valid will in order to save them legal or other difficulties when you think the patient is not of testamentary capacity. Above all, make notes immediately after you have seen the patient of the date and time you saw him, of his statements, and of the conclusion at which you arrived.

A BACILLARY DISEASE OF THE BLUE-TONGUED LIZARD (*TILIQUA SCINCOIDES*).

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(From the Baker Medical Research Institute.)

Foreword.

THE following study may appear to be remote from the domain of practical medicine, and therefore inappropriate for appearance in this journal. There is, however, ample justification for bringing to the notice of a medical audience any bacterium possessing a wide range of pathogenicity for the lower vertebrates. The "web of nature" is wide in its ramifications and every strand is important to the whole. I may, therefore, indicate the following possible points of contact between the present paper and the problems of practical medicine.

The bacillus to be described is highly lethal, either by injection or ingestion, to several widely different species of lizards, and to a less degree to frogs also. It is quite probable, then, that snakes too will be found to be vulnerable. I have not yet had opportunity to establish this experimentally, but, should it prove to be so, a means may be provided for devising a practical campaign of snake extermination in seriously infested areas. In this connexion it may be noted that Danysz introduced a virus for the extermination of rats, the effective organism being *Bacillus enteritidis* of Gaertner (see Muir and Ritchie's "Manual of Bacteriology", 1927, page 429). Danysz's method suffered, however, from the serious disadvantage that the potent bacterium was pathogenic also for domestic animals and for man. Should it prove feasible to employ the organism described in this paper as a snake exterminant, it is very unlikely that any such disadvantage would exist. I propose, when possible, to make further preliminary experiments regarding the pathogenicity of the bacillus for snakes; but I would be happy to supply cultures to any worker with facilities for the more extensive investigations which would be essential to demonstrate the possibility of a practical application of the kind suggested. Along with researches directed to the

production of successful therapeutic antivenins, there is room for work on the prophylaxis of snake bite, and any avenue of promise, such as the possible production of a snake virus, should be explored.

It is known that *Bacillus pseudotuberculosis rodentium* gives solid immunity in guinea-pigs against plague, while a vaccine of killed plague bacillus gives but a poor degree of immunity (see Ledingham's first Harbin Lecture, 1925, *Journal of State Medicine*, Volume XXXIV, No. 1). Again, the turtle strain of tubercle bacillus isolated by Friedmann was thought worthy of prolonged trials as an antigen in the prophylaxis and treatment of human tuberculosis, and, even at the present time, the value of such procedures and the relationship of this bacillus to the mammalian tubercle bacilli are questions which are still *sub judice*. (See Kolle and Wassermann's "*Handbuch der pathogenen Microorganismen*", 1928, Volume V, page 1052.) These facts illustrate that workers in human bacteriology and medicine cannot ignore the potentialities of bacteria which, while not directly concerned in producing human illnesses, are yet virulent for lower vertebrates. Such organisms may prove capable of yielding antigens of therapeutic value against other diseases.

Introduction.

During some dissections of *Tiliqua scincoides* in Tasmania in 1925, I encountered a specimen which presented granulomatous lesions of the peritoneum, ovaries and one hip joint. Both macroscopically and histologically these lesions resembled those of caseating tuberculosis in the human being, but no acid-fast bacilli could be found in sections stained by the Ziehl-Neelsen method. I proceeded no further with the study of the condition at that time, but in some subsequent experiments on *Tiliqua*, serosal and visceral nodules were seen in several specimens sent to me from Tasmania. Meanwhile, many dissections of Victorian specimens had been made without my seeing any trace of such lesions.

In subsequent investigations the disease proved indeed to be restricted to Tasmanian lizards, thirty-eight specimens of which all from districts around Launceston were examined. Of these thirty-eight lizards, seventeen exhibited spontaneous disease, an incidence of 45%. During the same period twenty-six Victorian specimens from various parts of the State were examined, and all were healthy. I have had no opportunity of examining lizards from other parts of Tasmania. From the spontaneous lesions of the North Tasmanian lizards a bacillus was isolated, fresh cultures of which, when inoculated into the cœlomic cavity or fed orally to healthy Victorian specimens, produced a rapidly fatal illness.

The investigation will be described in five sections as follows:

I Macroscopic Pathology of the Spontaneous Disease.
II Histology of the Spontaneous Disease.
III Bacteriology.
IV Inoculation and Ingestion Experiments.
V Summary.

I.—Macroscopical Pathology of the Spontaneous Disease.

Distribution of the Lesions.

The lesions observed in the seventeen spontaneously affected lizards were situated as indicated in the accompanying Table I.

Table I.

Organ.	No. of Lizard.																
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
Liver	−	+	+	+	±	+	+	+	−	+	+	+	+	+	+	+	−
Parietal serosa	+	+	+	+	+	−	−	−	−	+	−	+	−	−	−	−	−
Lungs	−	+	+	+	−	−	−	+	+	−	−	−	−	+	+	+	−
Pelvic tissues	−	+	+	−	−	−	−	−	−	+	−	−	−	−	−	−	−
Skeleton	+	−	+	−	−	−	−	−	−	−	−	+	−	−	−	−	−
Kidneys	−	+	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Ovaries	+	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Mediastinal tissues	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	+
Myocardium	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−
Spleen	−	−	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−

The liver was involved in all but three of the affected lizards. In three cases it was the only organ involved, and in several other animals (IV, V, VIII, X, XIV, and XV) it was the predominantly involved viscus, the nodules elsewhere being scanty. The hepatic lesions were in all instances of the same character, and were also representative of those in most other tissues (Figure I). They consisted of small well-defined nodules, situated either superficially or deep in the substance of the organ. The superficial nodules were sometimes sessile and subcapsular, sometimes prominently pedunculated. Their size varied from just visible dimensions up to two millimetres in diameter. Occasionally confluence of several contiguous nodules was observed. The smaller nodules were white and semitranslucent or opalescent in appearance, but the larger nodules often contained a yellowish centre which was visible through their white peripheral zone. In consistence the majority were firm, and some were quite hard and shotty in character. Their number varied from only three or four in the livers of the least affected lizards, up to perhaps sixty or eighty studding the most severely affected livers, as in numbers II and III.

The parietal serosa of seven of the diseased animals presented nodules, usually few in number. In several instances these were restricted to the pelvic region, involving the serous covering of the fatty bodies. In number II, however, the parietal nodules were numerous and widespread. In number III they were situated chiefly in the upper part of the cœlom around the pericardium and bases of the lungs. The nodules were identical in character with those already described in the liver, and were pedunculated more often than sessile.

The lungs exhibited pedunculated serosal nodules in eight lizards. These were numerous in number III, and few or solitary in the other specimens. In no case were any nodules found in the lung substance.

Three of the diseased lizards presented lesions in the pelvic connective tissues. In numbers II and X

these were single nodules attached to the external surface of the kidney capsule. In number III an irregularly lobulated fibrous mass five millimetres in diameter lay in the paracloacal tissues in the right side of the pelvic cavity.

The bones presented destructive lesions in three animals. In number III the heads of two ribs on the left side were enlarged and the costovertebral joints partly disorganized. In number XII the left clavicle and precoracoid were largely destroyed by an irregularly spherical mass 1·7 centimetres in diameter, which adhered slightly to but could be stripped from the subjacent pharyngeal muscles. The trachea was displaced to the right side, and the thoracic viscera were slightly compressed caudally and to the right (Figure II). The mass consisted

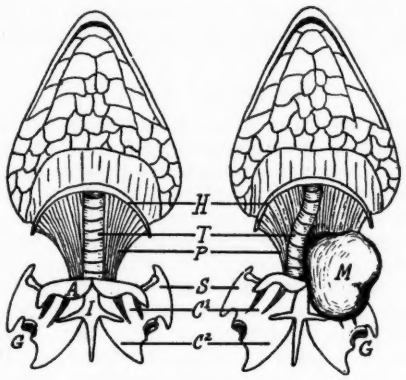


FIGURE II.
The mass on the pectoral girdle of number XII. (Half natural size.) The normal structure is shown on the left. *A* = clavicle. C^1 = precoracoid. C^2 = coracoid. *G* = glenoid cavity. *H* = hyoid. *I* = interclavicle. *M* = the mass. *P* = pharynx. *S* = scapula. *T* = trachea.

of a peripheral capsule of dense fibrous tissue with incorporated fragments of the destroyed bones and a central area of firm yellow degenerated material six millimetres in diameter. In lizard number I there was advanced disease of the head of the femur and of the acetabulum on the left side and the cavity of the disorganized joint contained some creamy yellow fluid (Figure III). The joint surfaces were totally destroyed by a carious process with the formation of many small sequestral fragments. The largest of these occupied a deep cavity in the ilium, which, in the dried specimen, formed a pin-hole perforation on the inner or pelvic aspect of the bone. New bone formation was evident at the periphery of the disorganized joint surfaces, especially at the cranial acetabular margin where a vertical flange of new bone rose from the ileo-pubic junction for a height of two millimetres. Similar bony outgrowths were present around the carious upper end of the femur. One cannot but be struck by the general similarity of this hip-joint disease to a tuberculous hip in a human being. Caries predominating over reparative bone proliferation, fragmentary sequestra, the destruction of the femoral head, and the presence of a central "cold abscess", are all features reminiscent of tuberculous arthritis in man.

FIGURE III.
The diseased left hip-joint of number I. (Natural size.) The healthy bones of the right side are shown for comparison.

The kidneys presented small nodules in two lizards. In number III there was a solitary subcapsular lesion, but in number II there were multiple nodules situated both subcapsularly and deep in the substance of both kidneys. These were similar to the hepatic and serosal nodules already described.

The ovaries were diseased in two animals. Figure IV indicates the appearance of these organs in number III. In addition to normal immature follicles, the right ovary contained one and the left ovary three large cystic masses filled with yellow fluid, and also several small superficial areas of old hæmorrhage. To casual inspection the cystic masses resembled half-matured, yolk-filled ova, but their contents were

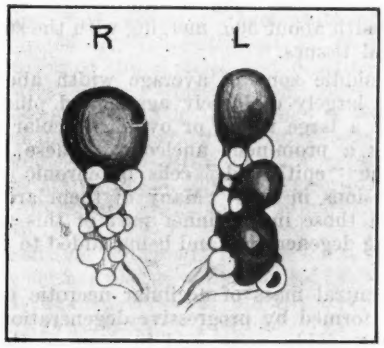


FIGURE IV.
The ovaries of number III. (Natural size.)

darker in colour than normal yolk, their inner surfaces somewhat ragged, and subsequent histological study fully established the pathological character of their walls. The ovaries of lizard number I (depicted in Figure V) were the seat of much more advanced disease. No trace of any healthy ovarian tissue was present, the organs being replaced by a pair of dark red-brown lobulated masses consisting of central pigmented degenerated material surrounded by a

zone of fibrous tissue of variable width. The left mass was irregularly ovoid and 18 by 13 millimetres in diameter, while the right mass was irregularly spherical and 13 millimetres in diameter.

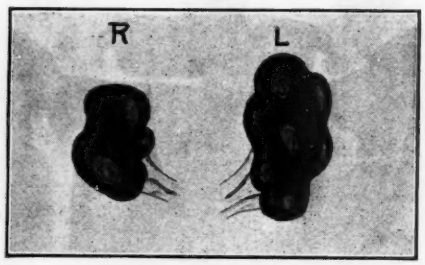


FIGURE V.
The ovaries of number I. (Natural size.)

The mediastinal tissues of number XVII presented a lobulated firm dark mass seven millimetres in diameter near the root of the right lung.

The ventricular myocardium of lizard number III contained two small nodules, and nodules were present also in this animal's spleen.

No lesions were found in the brain, adrenals, testes or musculature of any of the diseased animals.

II.—THE HISTOLOGY OF THE SPONTANEOUS DISEASE.

The Discrete Nodules in the Viscera.

The most frequent lesions, namely, the small discrete nodules in the liver, serous membranes, kidneys, myocardium and spleen, were similar in structure in all situations, and one description will suffice for all. Their structure is shown in Figures VI, VII and VIII. Each nodule consists of three distinct zones.

(*a*) A peripheral zone of fibroblastic tissue, of average width about 50μ, merging with the surrounding normal tissues.

(*b*) A middle zone of average width about 70μ, consisting largely of loosely aggregated plump cells each with a large round or ovoid vesicular nucleus containing a prominent nucleolus. These cells resemble the "epitheloid" cells of chronic granulomatous lesions in man. Many of them are vacuolated, and those in the inner part of this zone are undergoing degeneration and being added to the next zone.

(*c*) A central mass of acellular necrotic material, evidently formed by progressive degeneration of the cells of the middle zone, and in some of the larger nodules containing acicular spaces where fat and fatty acid crystals have been dissolved out in preparing the sections.

These characters indicate the decided chronicity of the lesions which is further suggested by the general paucity of leucocytes in and around the nodules. However, no example of complete quiescence or healing of a nodule has been observed, and abundant bacilli are to be found in all nodules stained for organisms. As will be described later, the bacilli are situated chiefly at the junction of the middle and central zones of the nodules.

Larger Granulomatous Masses in Soft Tissues.

The lobulated masses found in the pelvic and mediastinal tissues of lizards III and XVII respectively deserve special histological description. In parts of these masses small nodules similar to those just described are submerged. Elsewhere, larger irregular areas of degeneration are seen, each surrounded by a zone of tissue similar to the epithelioid cell zone of the small nodules. Besides the focal and irregular granulomatous areas, the masses under discussion present also areas of diffuse fibrosis with accumulations of small round cells (Figure IX). The paracloacal mass of lizard number III was evidently of long duration, for it consists chiefly of poorly cellular, partly hyalinized, fibrous tissue with patches of calcification. The mediastinal mass of number XVII, on the other hand, is richly cellular, contains much irregular degeneration, and presents all the features of an actively progressive inflammatory process.

The Diseased Bones.

The essential histological characters of the disease in the bones do not differ greatly from those already described. There is a progressive destruction of the involved bone and its replacement by an enlarging granulomatous mass consisting of the same three zones as appear in the visceral lesions. A peripheral fibrous zone of variable width acquires adhesions to adjacent structures and may contain unabsorbed spicules of bone and also areas of hæmorrhage and blood pigment. Internal to this lies a narrow epithelioid cell layer identical in structure with the middle zone of the visceral nodules. These two external zones form a capsule enclosing a cavity containing a solid or fluid mass of necrotic material which is about 50% soluble in ether and contains fatty acid crystals. If solid, as in the pectoral girdle lesion of lizard XII, this central degenerated mass presents concentric lamination. The histological details of bone proliferation around the diseased acetabulum and femoral head of lizard number I could not be determined, as the bones were preserved intact as a macroscopical specimen.

The Diseased Ovaries.

It was expected that microscopical study of the diseased areas in the ovaries would again disclose the characteristic granulomatous lesions. This, however, was not the case. The walls of the cystic masses in the ovaries of number III (Figure IV) consist of loose textured proliferating tissue merging with and evidently derived from the adjacent ovarian stroma, and containing much granular blood-pigment; while the fluid content of the cavities, unlike the yolk of adjacent healthy follicles, is much vacuolated and contains scattered pigment granules. Neither the peripheral tissue walls nor the central acellular contents of these cysts at all resemble those of the granulomatous lesions. The picture suggests rather the presence of retrogressive changes and yolk absorption in semi-matured follicles which have suffered some previous damage.

The ovaries of lizard number I (Figure V) present no residual healthy tissue whatever, but are replaced by masses of deeply pigmented degenerated material

enclosed within irregular fibrous walls. Histologically, while several small typical granulomatous nodules are present, the great bulk of the diseased tissue presents none of the characters of the granulomatous disease. Scattered through a richly cellular connective tissue are numerous large giant cells up to 100μ or more in diameter. These cells, which occur both singly and in groups, possess many nuclei situated chiefly peripherally, and their protoplasm contains large vacuolated spaces (Figures X and XI). Plump mononucleated cells bearing fat droplets are also present; and there are patches of old hæmorrhage and blood pigment.

From the foregoing descriptions it is clear either that the granulomatous lesions assume a greatly altered character in ovarian tissue, or else that the bulk of the ovarian disease is not directly due to the specific bacillary infection at all. It is here necessary to anticipate Section III, and to state that the characteristic bacilli could not be demonstrated in sections nor cultured from the fluid contents of the ovarian lesions described, so that these lesions cannot be regarded as merely structurally peculiar granulomata of bacillary origin. Nevertheless, since this ovarian disease occurred in two lizards with advanced bacillary lesions elsewhere, and as no similar ovarian disease has been seen in healthy lizards, it is to be assumed that the changes in the ovaries are related in some way to the bacillary infection of the animal. I believe the sequence of events to have been as follows. As the result of mild infection of the ovaries (several small granulomatous foci were found in the ovaries of number I), or possibly as the result of toxæmia only, maturing follicles were damaged and underwent retrogressive changes accompanied by disintegration and absorption of yolk and the occurrence of follicular hæmorrhages. The ovaries of number III (Figure IV) exhibit the early stages of this process restricted to the cavities of several separate half-matured follicles. In the ovaries of number I (Figure V) we see the late results, namely, absorbing hæmorrhages, phagocytosis of lipoids and pigments, and abundant formation of foreign-body giant cells containing large vacuoles of fat.

III.—Bacteriology.

Of the seventeen diseased lizards, the specific bacilli were found, either in smears or sections or by culture, in the lesions of fifteen. In two cases the organisms were not demonstrated—in number I, because the nature of the disease was not suspected at the time and no appropriate tissues were preserved, and in number XI, because the tissues were lost. In eleven lizards the bacilli were demonstrated in Giemsa-stained sections; in four in smears of the crushed nodules, and by cultures on blood agar in four cases. These last four lizards all yielded the same organism in pure culture.

Morphological and Staining Characters of the Bacillus.

The organism is a small bacillus of average length about 1·5μ and about 0·5μ in thickness. The length varies, however, the shortest individuals appearing as cocci, while the longest reach 6μ (Figure XII). The ends of the bacilli are rounded. Most of the organisms are separate, but diplo-bacillary forms and even short chains also occur. No capsules nor spores have been found.

Some bacilli stain uniformly, but many exhibit paler coloration centrally than at their ends. They are Gram-negative with Gram's and Jensen's stains, and non-acid-fast with Ziehl-Neelsen's method. Scanty acid-fast granules, found in primary cultures but absent from subcultures, are derived probably from the lizards' tissues. It may be noted here that acid-fast granules occur in both reptilian and human tissues (for example, the spleen).

In all but deteriorating cultures, the organisms are vigorously motile, hanging-drop preparations resembling, as Koch said of cholera vibrios, "a swarm of gnats". The speed varies, but under optimal conditions may reach 40μ per second, that is, 14 centimetres per hour. This velocity approaches that of the cholera vibrio which has a maximum rate of 18 centimetres per hour, and may be contrasted that that of *Bacillus typhosus* which moves at only 14 millimetres per hour (E. O. Jordon, "General Bacteriology", 1920, page 70). Special flagella stains reveal a single terminal flagellum of average length 6μ (Figure XIII).

Cultural Characters.

The organism grows readily on any of the ordinary solid media either at room temperature or at 37° C., blood agar being a particularly favourable medium. Clearly visible colonies appear around crushed fragments of the lesions of the spontaneous disease in eighteen to twenty-four hours. At room temperatures the cultures continue to increase for ten to fourteen days, then become stationary and undergo deterioration. The organisms remain viable for much longer, however, and healthy subcultures can be obtained from growths six or eight weeks old. The bacilli of old cultures exhibit excessive variations in size, impaired motility, fragmentation, and poor staining qualities. Incubation at 37° C. accelerates the evolution of the changes just described, so that the cultures become quiescent and deteriorated in five to seven days. The wide range of adaptability of the bacilli to temperature is doubtless related to the poikilothermic nature of their reptilian host. The times stated in the following descriptions of the growths on various media apply to cultures at room temperatures.

On blood agar the growth forms a moist translucent pale grey surface plaque of flat conical shape and with a sharply-defined even or slightly crenated edge (Figures XIV and XV). Frequently, radial striation or ribbing is present, but very moist cultures are quite even and smooth. The organism is hæmolytic and a clear zone of hæmolysis up to two millimetres or more in width is present in the medium beyond the edges of the colonies. In old cultures large granular deposits of blood pigment appear in the hæmolysed areas, and the whole medium becomes a murky brown colour. The cultures have a distinctive musty or sour odour, which is observable also on opening badly diseased or fatally inoculated lizards.

On plain agar the colonies are very similar but paler in colour and less luxuriant than on blood agar, and radial ribbing is inconspicuous. Like

that on blood agar, the growth is restricted to the surface of the medium.

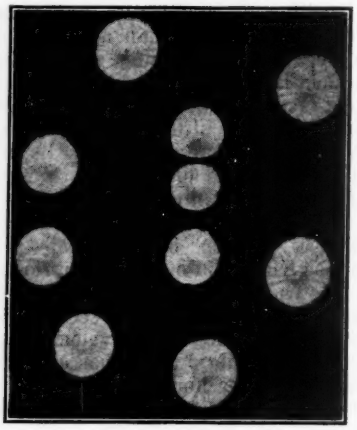


FIGURE XIV.
Colonies seven days old on blood-agar. (Natural size.)

On serum agar similar surface growth occurs, but the organisms penetrate also into the substance of the medium as a dense haze. Loeffler's serum medium also is penetrated by the growth and suffers fissuring and liquefaction. Peptone gelatine is speedily liquefied.

In stab cultures on any of the foregoing media the growth is most abundant at the surface, and is relatively scanty along the deeper parts of the needle track.

FIGURE XV.
Colonies of Figure XIV viewed by oblique illumination, showing the flat conical shape and radial ribbing. (Twice natural size.)

Cultures on egg media (Dorset's and Hohn's) are very characteristic. The early growth appears as a moist granular surface film of pale yellow-pink colour. As this extends it often deepens in colour to a bright orange. Well-established cultures have a penetrating "cheesy" odour decidedly different from that of cultures on the agar media. After three or four days the colonies frequently develop small surface bubbles of gas or even large bullæ, which rupture, leaving crater-like impressions on the surface of the growth. The degree of this bubble formation varies from tube to tube of the egg media, and its occurrence appears to depend partly on the moistness of the medium. I have not been able to ascertain the composition of the gas produced. Ordinary tests for hydrogen sulphide (with metallic salt solutions) and for carbon dioxide (with lime water) give no decisive results; and, as far as can be determined with the small quantities of the gas available, it is not inflammable, and therefore contains no great quantity of either gaseous hydrocarbons or hydrogen. Simultaneously with the development of gas, the egg media commence to undergo fissuring, and eventually total disintegration and liquefaction take place.

In peptone water, plain broth, and Hartley's tryptic broth, the organisms grow freely, a minute inoculum producing visible opalescence in twelve hours and dense uniform turbidity within forty-eight hours. Tryptic broth appears to be the most favourable liquid medium. In litmus-milk growth is relatively slow, and no acid production nor clotting appears. The milk suffers alteration, however, for in four or five days the casein sinks as a slimy precipitate poor in bacilli, the fat floats at the surface, while the majority of the organisms are found in the intervening whey.

In a McKintosh-Fildes jar no growth occurs on blood agar even after seven days' incubation either at room temperature or at 37° C.; the organism is an obligatory aerobe. Anaerobic conditions for this period do not injure the bacilli, however; for, on returning the culture tubes to the air, vigorous growth promptly ensues.

Fermentative Properties.

The bacillus possesses a wide range of fermentative powers. Table II gives the results with various substances in peptone water at room temperature (about 20° C.). An indication of the relative amounts of acid and of gas produced from the various substances is given.

The greatest production of gas occurs with maltose and glycerine, though the latter exhibits initial slowness in this respect. The fermentations produced by organisms obtained from three different primary cultures were tested, and were essentially similar in all.

From the foregoing results and from the destructive changes produced in serum, gelatine and egg media, it is evident that the organism possesses powerful and diverse lytic powers.

Situation of the Bacilli in the Lesions of the Spontaneous Disease.

In eleven of the diseased lizards, sections of various lesions were stained for organisms by Giemsa's method. In all cases abundant bacilli were found to be present. They were most numerous in the recently necrotic material just internal to the epithelioid-cell zone, and relatively few and scattered in the more central degenerated material (Figure XVI). None were found in the epithelioid cell zone

itself. Apparently, then, the tissue reaction of which this zone is the expression is excited largely by diffusible toxins liberated from the colony of bacilli in the adjacent necrotic zone.

TABLE II.

Medium.		Twenty-four Hours.	Forty-eight Hours.	Ninety-six Hours.
Pentoses	Arabinose ..	—	—	—
	Xylose ..	—	—	—
Hexoses	Dextrose ..	A.[1]	A.	A.
	Lævulose ..	A.	A.G.[2] (sl.)	A.G.
	Mannose ..	A.	A.G. (sl.)	A.G.
	Galactose ..	A. (sl.)	A.G. (sl.)	A.G. (sl.)
Disaccharides	Maltose ..	A.	A.G.	A.G. (abundant)
	Lactose ..	—	—	—
	Saccharose ..	A.	A.	A.
Trisaccharide	Raffinose ..	—	—	—
Polysaccharides	Dextrin ..	A.	A.	A.G. (sl.)
	Inulin ..	—	—	—
Glucosides	Salicin ..	—	A. (sl.)	A.
	Amygdalin ..	A. (sl.)	A. (sl.)	A. (sl.)
	Glycerol ..	A. (sl.)	A.G. (sl.)	A.G. (abundant)
Polyhydric Alcohols	Erythritol ..	—	—	—
	Adonitol ..	—	—	—
	Mannitol ..	A.	A.G. (sl.)	A.G.
	Sorbitol ..	—	—	—
	Dulcitol ..	—	—	—
	Inositol ..	—	—	—

[1] Acid. [2] Acid and gas.

IV.—Inoculation and Ingestion Experiments.

Experiments with Blue-tongued Lizards.

As stated already in the introduction, only robust Victorian specimens were used for transmission experiments.

Six adult lizards were given intracœlomic injections of fresh subcultures suspended in sterile water. The doses used and the results obtained are given in Table III. (The doses stated are only approximate; they were estimated by naked-eye comparison of the opalescence of suspensions of the bacilli with that of standard vaccines of *Bacillus coli communis*, an organism of similar size and shape, the smaller doses then being obtained by dilution.)

TABLE III.

Lizard Number.	Dose of Bacilli.	Result.
I	6,000 million	Died in 61 hours.
II	6,000 ,,	Died in 71 hours.
III	1,000 ,,	Died in 72 hours.
IV	200 ,,	Died in 84 hours.
V	40 ,,	Died in 26 days.
VI	10 ,,	Died in 16 days.

The following additional experiments also were made.

Two lizards were given several minims of a cloudy suspension of bacilli by mouth; both animals died, one in eighty-seven hours and the other in ninety-six hours. Lastly, a nodule of the spontaneous disease from lizard number VI of Section I was ground up in sterile water and injected into the cœlom of a healthy young Victorian specimen. This animal died in one hundred and fourteen hours.

In the foregoing transmission experiments the specific bacillus was recovered in pure culture from both the cœlomic fluid and the heart blood of all nine infected lizards. Koch's fourth postulate has therefore been amply satisfied.

The Pathological Findings in the Experimentally Infected Lizards.

In the seven animals which succumbed within a few days, closely similar changes were found, namely, intense hyperæmia of the viscera and parietal serosa, a moderate amount of viscid or hæmorrhagic exudate in the cœlomic cavity, and easily detached flakes of yellow fibrinous deposit on the visceral surfaces. In several instances small interstitial hæmorrhages were present in viscera, particularly the liver, kidneys and lungs. When opened, the lizards all emitted a characteristic penetrating sour smell very similar to that already noted in describing cultures of the organisms. In all cases smears of the cœlomic exudate contained numerous bacilli, as did also films of the heart blood (Figure XVII). Sections of the viscera contained enormous numbers of bacilli, which were most numerous in the liver and spleen (Figure XVIII). It is clear that these lizards died from an overwhelming septicæmia. For a cold-blooded animal, an illness which is fatal in two to five days is, indeed, a fulminating one.

The two animals which survived longer, one for sixteen days and the other for twenty-six days, exhibited lesions more comparable with those of the spontaneous disease. In both there were several large yellow grey masses up to one centimetre in diameter in the substance of the liver, smaller scattered soft nodules on the parietal serosa, and patchy recent adhesions between the liver and parietes. In addition there were present general congestion and œdema of the viscera, and turbid cœlomic exudate. Many bacilli were present in the tissues and blood. The focal lesions in the liver and elsewhere, though much more acute in character than those of the spontaneous disease, exhibited a recognizably similar histological structure. Surrounding massive areas of necrosis were richly cellular zones of the characteristic epithelioid cells, but as would be expected, there was no peripheral zone of fibrosis. Figure XIX of one of the smaller nodules in the liver shows the general similarity to the spontaneous lesions depicted in Figures VII and VIII.

The history of the illness of the longest surviving lizard is of some interest.

Within three days of receiving the injection (forty million bacilli), this lizard, like those which succumbed at this stage, appeared to be sluggish and almost moribund. He remained very ill for several days, but on the seventh day was clearly better. Thereafter, though not as lively as a healthy animal, he appeared relatively well until his death on the twenty-sixth day.

Probably the early phase of severe illness on the third to the sixth day was occasioned by a septicæmia similar to but less severe than that which proved fatal at this stage in the other animals. This lizard, however, recovered from the early blood infection, to die later from the results of massively established lesions in the viscera.

Inoculation Experiments with Other Lizards.

Seven individuals of a species of small gecko (*Gehyra variegata*) were given intracœlomic inoculations with the results indicated in Table IV.

In all seven animals there was a moderate amount of sticky cœlomic exudate, the liver was pale and mottled, the characteristic smell noted in the blue-tongued lizards was present, and the bacilli were present in enormous numbers in the exudate, viscera and blood. An eighth gecko was given one minim of a

TABLE IV.

Number.	Dose.	Result.
I	250 million bacilli	Died in 24 hours.
II	250 " "	Died in 30 hours.
III	250 " "	Died in 32 hours.
IV	250 " "	Died in 32 hours.
V	250 " "	Died in 56 hours.
VI	2 " "	Died in 137 hours.
VII	1 " "	Died in 126 hours.

faintly opalescent suspension of bacilli by mouth. It died in forty-eight hours.

One specimen of the Jew or bearded lizard (*Amphibolurus barbatus*) was given one thousand million bacilli by intracœlomic injection. It died in fifty-one hours, and presented general visceral hyperæmia, and profuse bloody exudate in the body cavity. The organisms were recovered in pure culture from both the exudate and the heart blood.

Inoculation Experiments with Frogs.

Two frogs (*Limnodynastes dorsalis*) received each five hundred million bacilli by intracœlomic injection. One of the animals died in ninety-six hours and exhibited a generalized serositis with viscid exudate and flakes of fibrin on the viscera, intense congestion of the liver and spleen, and numerous bacilli in the heart blood.

The second frog was sluggish and almost moribund for three days, but then gradually improved and became apparently quite well within a week. It was killed on the eighteenth day and no relevant abnormality was discovered, microscopical sections of the viscera showed no granulomatous lesions and no bacilli could be found.

This observation is of interest, suggesting that, while frogs may be killed by an initial acute septicæmia, if they survive this, they are not subject to the later development of inflammatory foci in the viscera.

Inoculation of Guinea-pigs.

Four guinea-pigs were inoculated subcutaneously each with one thousand five hundred million bacilli. At the site of injection in each case there appeared a small discharging sore, which, however, healed completely within two weeks, leaving no residual thickening nor local glandular enlargement. Smears of the discharge contained a few poorly stained bacilli only, and cultures at room temperature produced no growth. Two of these guinea-pigs were killed on the twenty-first day after injection, but no local nor systemic evidence of bacillary infection could be found. The remaining two guinea-pigs were killed in five weeks from the date of injection and these also exhibited no sign of bacillary disease.

Two more guinea-pigs were inoculated intraperitoneally each with two thousand million bacilli from a culture which had been grown at 37° C. for three days. Both animals remained quite well. One was killed in three weeks and one in eight weeks, and all organs were completely normal.

It may be concluded then that the organism is non-pathogenic for guinea-pigs. I have not experimented with other animals.

Immunity Phenomena Induced in Lizards by Non-lethal Inoculation.

The quite apparent chronicity of the lesions in many of the spontaneously diseased lizards, although these lesions contain abundant bacilli, suggests the possible existence of some degree of immunization in these animals. In view of the vigorous motility of the bacilli, it seemed not unlikely that such immunization, if present, might include the development of specific agglutinins.

Accordingly some blood serum from several of the fatally inoculated lizards was mixed with suspensions of actively motile bacilli, and the mixtures examined for several hours under the microscope. On each occasion there was observed a very striking and immediate acceleration of the bacillary movements as compared with serum-free controls, the organisms darting about with dazzling speed hither and thither. This acceleration, however, was only temporary, the bacillary movements subsiding to normal speeds within thirty to forty-five minutes. Although watched for several hours longer, the preparations exhibited no evidence of further loss of motility nor of agglutination.

These observations, however, performed with the blood of animals dead of a speedily fatal septicæmic infection, are of little value in the search for possible immunity phenomena, and the following experiment was performed in an endeavour to obtain immunization by a non-lethal inoculation with organisms of diminished virulence.

A large robust Tasmanian blue-tongued lizard was used. A preliminary test was carried out, blood obtained by snipping off the tip of the animal's tail being used. This blood produced no agglutination of a fresh suspension of living bacilli, at a dilution of approximately one in ten, and observed for several hours at room temperature. The lizard was then given an intracœlomic injection of two million bacilli from a poorly motile, three-weeks old culture on Hohn's egg medium. The lizard remained quite well, at no time developing any of the signs of septicæmic illness, such as were observed in fatally inoculated specimens. The animal was killed by chloroform on the fortieth day following the single inoculation, some blood was aspirated from the inferior *vena cava*, and the serum allowed to separate. This was mixed with fresh motile bacillary suspensions in plain water in dilutions of one in twenty, one in sixty, one in one hundred and twenty, one in two hundred and forty, one in five hundred, and one in one thousand, and the preparations were observed both macroscopically and microscopically for twenty-four hours at room temperature about 15° C. In the one in twenty dilution, commencing agglutination was apparent microscopically in thirty minutes; in forty-five minutes many large clumps had formed, but there were still numerous freely motile bacilli present, while in seventy-five minutes the majority of the organisms were clumped and only occasional free individuals were to be seen. In the one in sixty dilution, early agglutination was seen in forty-five minutes; in seventy-five minutes many moderate sized clumps had formed, but many bacilli still remained free, while subsequently little further change occurred, many free organisms being still in evidence several hours later. In the one in one hundred and twenty dilution slight agglutination was seen in seventy-five minutes, but no appreciable change thereafter, the majority of the organisms remaining free. In the one in two hundred and forty dilution a few small clumps had formed in ninety minutes, but there was little subsequent change. Macroscopically, in the one in twenty dilution, early flocculation was apparent in two hours and complete precipitation had occurred in eighteen hours. In the one in sixty dilution nearly complete precipitation occurred within eighteen hours, but a very faint haze of residual motile bacilli remained in the suspension fluid. Little or no macroscopical change was seen in the higher dilutions.

Several features observed during the actual process of agglutination are of interest. The earliest indication of this change was the presence here and there of individual bacilli which, instead of exhibiting the normal free translatory movement, appeared to be anchored by their flagella and to be struggling this way and that in an ineffectual endeavour to escape. Organisms in a similar plight then became arrested alongside each other, and thus formed the nucleus of an agglutinated clump, which steadily enlarged by the addition of further bacilli at the periphery. While the bacilli massed together in the centres of the clumps were completely stationary, those at the periphery often continued to exhibit struggling movements for a considerable period. Such bacilli appeared each to be held by the extremity of its single flagellum as by a leash, the body of the organism darting and swinging freely around this point of fixation.

ILLUSTRATIONS TO THE ARTICLE BY Dr. RUPERT A. WILLIS.

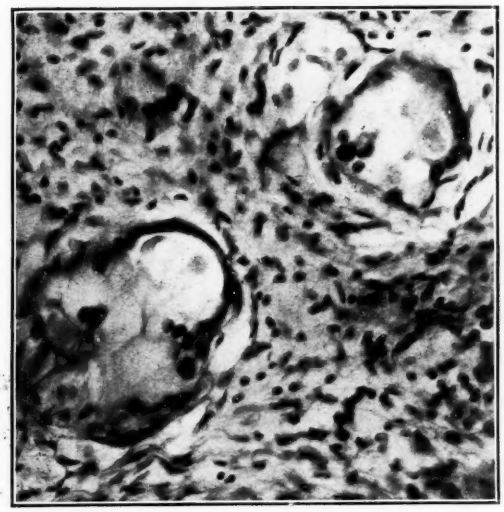


Figure XI.

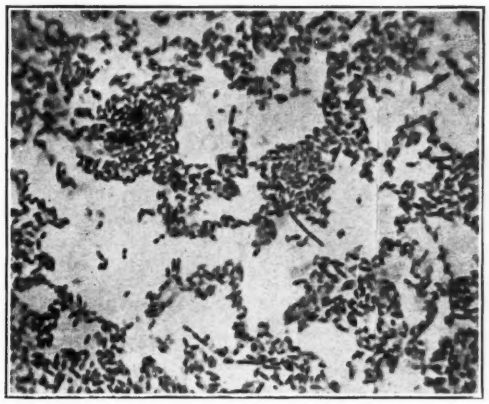
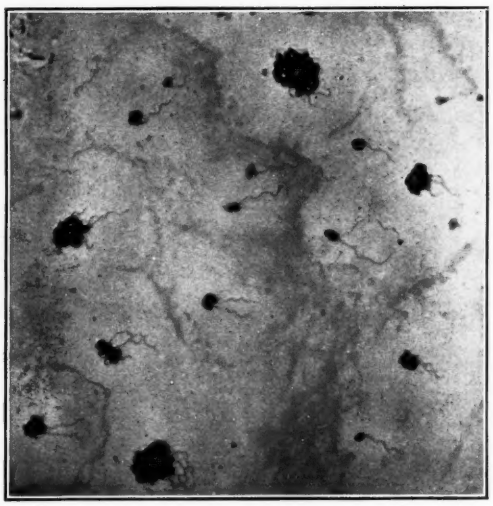


Figure XII.



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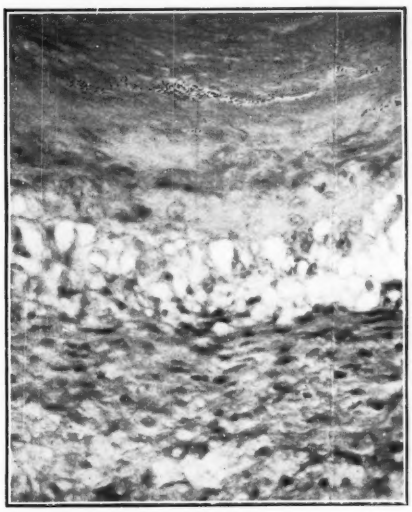

Figure XVI.

Figure XVII.

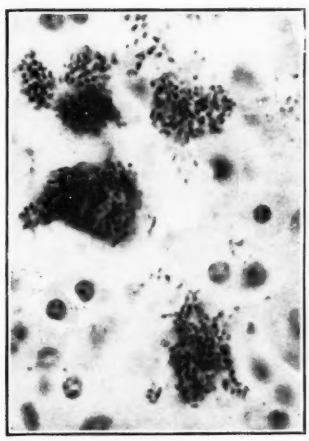

Figure XVIII.

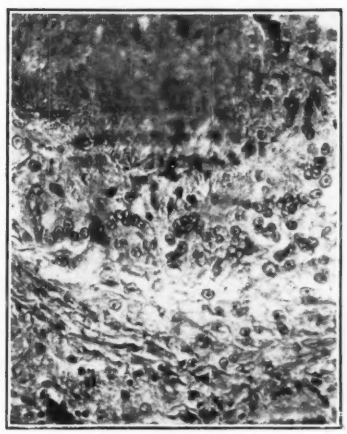

Figure XIX.

ILLUSTRATIONS TO THE ARTICLE BY DR. RUPERT A. WILLIS.

FIGURE I.

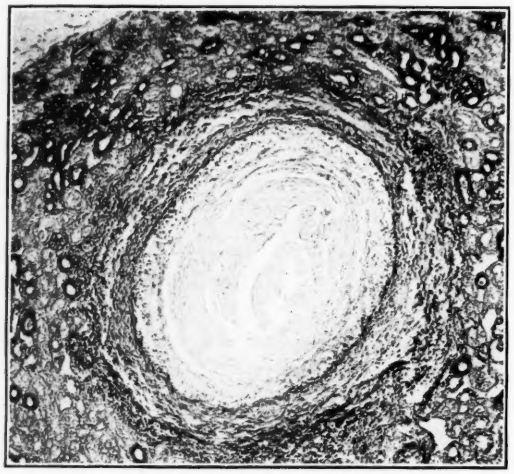

FIGURE VI.

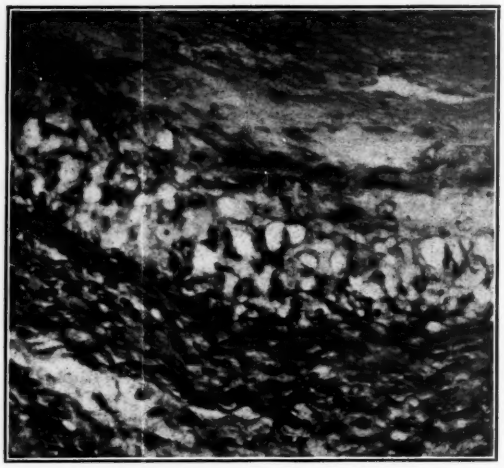

FIGURE VII.

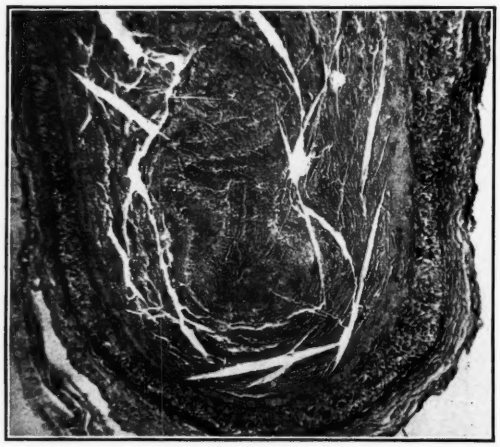

FIGURE VIII.

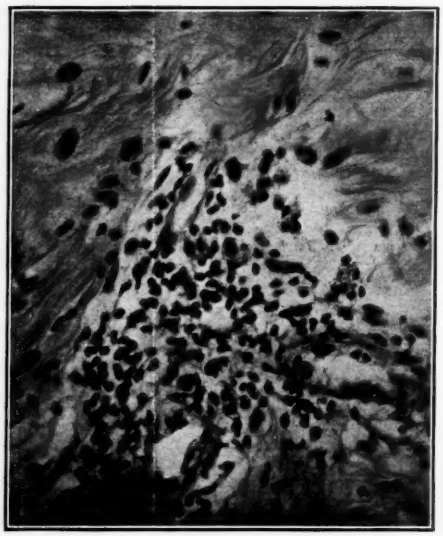

FIGURE IX.

FIGURE X.

All the viscera of the lizard used in the foregoing experiment appeared quite healthy; no sign of any granulomatous nodules or other lesions could be discovered. Hence, the small dose of bacilli from an old culture, though potent as an antigen in provoking agglutinin formation, was ineffective in establishing the specific lesions of the disease. This phenomenon is precisely similar to those observed in many human infections, and points to the probability that lizards may be successfully immunized against infection by the bacillus. I have not yet had an opportunity of carrying out further experiments on this point.

V.—Summary.

An endemic infective disease exists among blue-tongued lizards (*Tiliqua scincoides*).

The lesions of this disease have the features of chronic granulomata and in many ways resemble, both macroscopically and microscopically, tuberculous nodules in mammals. Each lesion consists of a central necrotic mass surrounded by a reactive epithelioid-cell zone peripheral to which is a zone of fibrosis.

The causative organism is a small, Gram-negative, non-acid fast, vigorously motile bacillus, with a single terminal flagellum. It grows freely on nearly all ordinary media, either at room temperature or 37° C., but is a strict aerobe. It possesses a wide range of fermentative powers.

Intracœlomic injection or oral administration of adequate numbers of these bacilli to healthy blue-tongue lizards produces a fatal illness, and in the lizards which survive for two weeks or longer visceral lesions resembling, though more acute than, those of the spontaneous disease are found.

A limited number of experiments on other species indicate that the bacillus probably possesses a wide range of pathogenicity amongst lizards, and this pathogenicity extends also to frogs. Guinea-pigs, however, are not affected.

Non-lethal inoculation of healthy lizards with bacilli of impaired virulence excites the formation of potent agglutinins in the blood serum, though failing to produce actual infection.

Acknowledgements.

It is a pleasure to record my thanks to Miss J. Somerville, of Launceston, who collected and sent me all the Tasmanian lizards referred to in this paper, thirty-eight in all. My wife has been my encouraging assistant throughout the investigation, caring for the captive animals, aiding in the bacteriological work, and preparing all the microscopical sections, about eighty in number. I am indebted to Dr. W. J. Penfold, Director of this Institute, for his stimulating interest and advice.

Legends to Illustrations.

Figure I.—Examples of the nodules in and on the liver, some discrete, some confluent. (Natural size.)

Figure VI.—A nodule in the kidney of number II, showing the three zones. (× 60.)

Figure VII.—High-power view of part of Figure VI, showing the structure of the three zones. (× 500.)

Figure VIII.—A serosal nodule, showing acicular spaces in the degenerated zone from which fatty-acid crystals have been dissolved. (× 75.)

Figure IX.—From the pelvic mass of number III, showing hyaline fibrous tissue and a collection of small round cells. (× 500.)

Figure X.—From the left ovary of number I, showing many giant-cells in a richly cellular tissue. (× 60.)

Figure XI.—High-power view of part of Figure X. (× 360.)

Figure XII.—A pure culture of the bacilli, stained with fuchsine. (× 1250.)

Figure XIII.—The bacilli stained for flagella by the method of Plimmer and Paine (*Journal of Pathology and Bacteriology*, Volume XXIV, 1921). (× 1250.)

Figure XVI.—A Giemsa-stained view of Figure VII, showing the situation of the bacilli at the periphery of the central necrotic zone. (Retouched.)

Figure XVII.—A Giemsa-stained film of the heart blood of a fatally inoculated lizard, showing bacilli more numerous than the blood corpuscles. (× 1250.)

Figure XVIII.—A Giemsa-stained section of the spleen from the same animal as Figure XVII, showing dense masses of bacilli. (× 1250.)

Figure XIX.—From the experimental lesions in the lizard which lived for twenty-six days after inoculation; showing the similarity to the lesions of the spontaneous disease. Compare with Figures VI, VII, VIII. (× 300.)

INJURIES AND DISEASES IN AUSTRALIA ATTRIBUTABLE TO ANIMALS (OTHER THAN INSECTS).[1]

Series IV.

By J. Burton Cleland, M.D.,
Marks Professor of Pathology, University of Adelaide.

In the *Australasian Medical Gazette* of September 14 and 21, 1912, the first series of reports of injuries and diseases in Australia attributed to animals, other than insects, was published. In 1915 this was supplemented by a further collection of cases published in the Sixth Report of the Government Bureau of Microbiology, Department of Public Health, New South Wales (page 270). In 1924 a further paper on the subject appeared in The Medical Journal of Australia, October 4, 1924. The present communication represents cases collected since the latter date.

Injuries from Man.

Dr. John MacPherson(1) has recently referred to poisoned spears in use at one time by Australian natives. It would appear that in the New England district a definite poisonous substance, probably with a scent, was employed. In North Queensland the acrid milky juice of the milky mangrove (*Excœcaria agallocha* L.) was used. In North Australia spears made of red mangrove wood (*Bruguiera rheelii* Blume) were by their nature liable to cause ugly festering wounds. He mentions the Reverend George Taplin's description of poisoning spear-heads and other objects used on the lower Murray by steeping them in putrefying corpses, and Dr. Herbert Basedow's account of inserting the vertebra of a large fish into decaying flesh and then tying it to the head of a fighting spear.

Injuries from Other Mammals.

Camels.—Dr. J. H. Saunders has told me of the following case.

On going to Moora, Western Australia, as Acting Government Medical Officer about 1913, he found an Afrikander who three days previously had gone to bed as the result of injuries received by the bite of a camel. He had apparently ridden a bull camel in the "must" state with-

[1] Read at a meeting of the South Australian Branch of the British Medical Association on July 30, 1931.

out crossing the reins under its chin. The camel bent its head round and bit him to the bone on the left tibia, shook him like a rat and threw him by dragging him to the ground. There was marked emphysematous gas crepitation and the appearance of gangrene. He was immediately sent to Perth for amputation and recovered with the loss of his left leg.

In August, 1923, a man, aged twenty-nine, living at Cockburn, was admitted to the Broken Hill Hospital, his foot having been bitten near the ankle by a camel when he was putting on harness. The camel had seized him by the foot and had thrown him for yards. The two upper teeth penetrated into the bones of the ankle and the lower jaw met with the sole of the boot. The foot was a good deal bruised with several teeth marks extending deeply in. The temperature became raised and the foot a good deal swollen round the ankle. Two days later the swelling was more extensive and several deep incisions were made which let out a considerable amount of pus. Three days later again incisions were made in fresh places and more pus evacuated. The ankle joint is said to have been opened surgically. In six weeks the wound had healed and the patient was walking well.

Pig Bite.—Dr. Brynmor Jones, of Mount Pleasant, South Australia, has kindly given me notes of a bite from a pig received in August, 1925.

The man was killing a pig and had already cut its throat when the pig snapped at him, completely severing the terminal phalanx of the right middle finger, the severed segment falling to the ground. Dr. Gilbert Jose treated the patient at Woodside, cleaned the wound and inserted stitches, but these had to be removed on account of sepsis. The finger had to be incised several times and treated with a constant eusol bath.

Injury from a Sea-Leopard (Hydrarga leptonyx de Blainville).—Dr. Eric Jeffrey, of Hobart, has informed me of an injury caused by a sea leopard.

In November, 1930, two men were fishing from a dinghy at Prince of Wales Bay, about four miles upstream from Hobart. Some fish were on the floor of the boat. Suddenly a large animal (probably a sea-leopard in quest of a fish) heaved itself up on to the side of the boat. One man fell overboard. He says he was dragged out of the boat by the beast. Possibly he was jolted out and bitten by the beast in his panic. He was taken to the Hobart Public Hospital with wounds in his calf made by an animal with large teeth. His description, Dr. Jeffrey says, would seem to apply to a sea-leopard. One had been seen on the shore near by in the previous year.

Dr. Jeffrey adds an amusing anecdote. Some twenty odd years ago a constable went to arrest a drunken man who was lying one night in a gutter near the Hobart wharves. He ended by shooting the "drunk", which—an adult sea-leopard—is now in the Hobart Museum.

Injury from a Dingo.—The daily Press a few years ago reported the following incident.

A young farmer, hearing his cow bellowing, took a lantern and went to investigate. He found her trying to beat off a dingo which persistently returned to a young calf born that night. The farmer stooped to pick up the calf when the dingo sprang at him and he lost his balance. He was badly damaged before he could beat it off.

In another instance a child left sleeping under a tree while the mother was helping her husband to burn off, was being dragged away by a dingo when the child's cry attracted the parents.

Injury from a Fox.—Dr. A. Chenery, of Wentworth, New South Wales, has kindly supplied me, through Mr. J. Sutton, with the following account of injuries received from a fox.

Mr. L. McN., of Bunnerungie Station, near Lake Victoria Station, was sitting down behind some blue bush at the side of a shallow gully and was imitating the squeal of a rabbit with a piece of tin between his teeth (and a very realistic squeal it is when done properly). He had on an old dark grey felt hat, when the fox came behind and above him through the blue bush. The slight noise it made caused him to half turn his head, and just as he did so the animal jumped at what evidently he thought was the rabbit, and grabbed Mr. McN. by the cheek. The impact half knocked him over and the fox disappeared hurriedly on discovering its mistake. The patient came in forty miles to see Dr. Chenery and have his wound dressed. There were two or three small lacerations on his right cheek and one on the side of his nose. These looked like teeth marks.

Feral Domestic Cat.—E. A. Le Souef,[2] Director of the Zoological Gardens at South Perth, makes the following statement.

As an example of how cats are developing in Western Australia, there is an exhibit of a very large one in the Perth Museum that attacked two children, but was fortunately killed by their father with an axe.

Injuries from an Old Man Kangaroo.—In February, 1929, a newspaper made the following report.

A man at Dubbo, an ex-boxer, feeding the animals in the park, had been attacked by a big old man kangaroo and had to use his fists, making his opponent's nose bleed. Eventually they fell together, and the man was bitten and scratched before a youth could drive off the kangaroo. Dr. Flower, of Dubbo, has kindly made inquiries for me, but cannot find any trace of the man concerned, so evidently the injuries received must have been trivial.

Wombat Bite.—Charles Barrett[3] mentions that a young naturalist, in capturing a wombat desired as a pet, was bitten on the knee and suffered for weeks afterwards.

Injuries from the Spur of the Platypus.—Dr. Frank Tidswell,[4] in collaboration with Dr. (now Sir Charles) Martin, has described the effects of the poison associated with the spur on the hind foot of the platypus and summarized the literature up to 1906. Dr. W. Kent Hughes[5] has recently described a case of injury from this cause.

A lad fishing in the Yarra had caught a platypus with a bait. In removing the hook the animal dug its hind claws into his hand; twenty minutes later the hand was very swollen, tense and hot, with some swelling extending to the elbow and some pain in the axilla. He stated that directly after the puncture his hand had become numb in the vicinity of the puncture. There had been no bleeding. Next morning there was still some swelling present. He had had a restless night, and in the morning complained of a dull pain in the epigastric region. His condition when first seen had been quite alarming.

The same author later[6] records a description, in 1874, by the late J. W. Rusden, of the effects on a native who was wounded on the wrist by the hind feet of a dying platypus. Within an hour there was swelling around the puncture marks. The arm became much swollen, especially at the shoulder, and the "swelling of the gland" was so painful that the arm was useless for some time.

Injuries from Birds.

Australian Magpie.—Dr. R. H. Pulleine tells me that he knows of a case in which a patient's eye had been injured by a magpie (*Gymnorhina*) and a traumatic cataract had resulted.

Attacks by Wedge-Tailed Eagles (Uroætus audax).—The Perth daily Press of February 11, 1926, contains the following sensational story:

An eagle fiercely attacked a sleeping child who had been left on a bed just inside the window. The child's screams brought the mother to its assistance. The eagle

had buried its talons into the bed-clothes, but the mother diverted its attention sufficiently long to enable her to snatch the baby away before it was injured. A man who was passing saw the eagle enter by the window and rushed in to help. The eagle flew up to the ceiling and swung down upon the man again and again, flapping its wings about him and pecking at his face and head. He finally overcame it.

Mr. F. Lawson Whitlock[6] has made careful inquiries into the authenticity of this account and finds that there is no evidence of a child having been attacked at all.

The incident occurred at a settler's cottage in forest country near Manjinup, situated between Perth and the South Coast. The owner of the cottage kept a flock of tame pigeons which appear to have attracted the eagle. As a little girl was crossing the adjacent clearing the eagle made a swoop after her, following her right up to the veranda as she ran screaming into the house. The eagle perched on the sill of the veranda in which an infant happened to be asleep. A man near by let fall a flap shutter, thus making the eagle a prisoner in the veranda. After a scuffle it was caught by means of a chaff-bag, but neither the infant nor the little girl received any injury.

Mr. Whitlock was able to collect another account of an attack by an eagle, communicated by Mr. A. Lawrence, of Young's Siding, near Albany.

A little boy, aged six, was playing on the flats near the house when the inmates were alarmed by his terrible screams. Rushing out, they found an eagle attacking the child. Their loud shouts drove the bird away, but so deliberate was it that it only retired to the top of a broken gum. The child escaped any physical injury, but the sight of any large bird of prey passing over still sends him running into the house in alarm. He was wearing a dark blue jersey inconspicuously piped with red; he wore no shoes or stockings and no cap, and has a mop of bright curly hair. It is thought that the bright hair may have attracted the eagle to the boy.

Mr. Whitlock himself has stood in the nests of eagles and handled their young, but has never been attacked by the old birds, though they flew round at a safe distance, uttering yelping cries.

Injury from a Cassowary.—Charles Barrett[7] states that the following account of an injury by a cassowary (*Casuarius casuarius* L.) was vouched for by several persons.

A party of boys in the Daintree district in North Queensland put up a cassowary which suddenly turned in its race from the yelping dogs. The boys turned too, but one stumbled and fell. The cassowary, charging wildly, struck by intention or by chance the lad as it rushed over him and ripped his neck with a claw. The wound was fatal.

Basal-Celled Epithelioma of the Finger in the Scar from a Bite of a Kookaburra.—Dr. N. Paul,[8] in giving examples of rodent ulcers probably due to trauma or contributed to by such, mentions one that arose at the site of an injury inflicted by a laughing jackass (*Dacelo gigas*).

Injuries from Reptiles.

The large Australian literature on snake-bite and snake venom has not been included in any of these series.

Crocodile.—I am indebted to Dr. A. M. Langan, Medical Superintendent, Cairns Hospital Board, for the following information about a man who was attacked by a crocodile (so-called "alligator") at Cairns in January, 1929.

The person in question, a man aged twenty-five years, was bathing at night with several friends. There was a faint light from an electric street lamp a short distance away. His friends were sitting on the shore and he was squatting in about two feet of water when something suddenly struck him on the back. He promptly took to his heels. His friends and other eye-witnesses state that there is no doubt that he was attacked by a crocodile, and affirm that they were able to see it distinctly. He was brought to the Cairns District Hospital, where he was examined by me. He had a semi-circular wound which almost looked as if it had been made with a scalpel. It was little more than a scratch at each end, and gradually became deeper towards the central gap until it just entered the superficial fascia. I presume it was inflicted with the saurian's claw. I cleaned it out with ether and iodine under a general anæsthetic and inserted about twenty-five silkworm gut stitches. The sutured wound was fomented every four hours with saline foments and healed remarkably well. He also had a lacerated wound about two and a half inches long on the outer side of the right buttock. This extended for a distance of about two inches into the gluteal muscle. I cleaned the wound in the same manner and stitched it up, with drainage, but it broke down and healed by second intention. This wound, I think was probably made by a canine tooth.

A newspaper reference later in the year referred to articles belonging to a Chinese being found inside a crocodile in North Queensland, thus explaining the disappearance of the Chinese.

Alleged Dangerous Lizards.—In the *Journal of the Royal Society of Western Australia*,[9] for the session 1927-1928, the following note appears:

"Mr. Glauert exhibited specimens of gecko lizards, *Gymnodactylus miliusii* and *Nephrurus asper*. The latter, a dangerous species, apparently imitated the former."

In reply to an inquiry from me, Mr. Glauert has informed me that owing to undue condensation a wrong interpretation had been placed on his statement. He had said that "the latter species was reputed to be dangerous and feared by the blacks for reasons which up to the present it has not been possible to determine". In his letter he adds that "the aborigines are generally considered to be accurate in their opinions of animals, and I would like to discover why they dread this little lizard and the little black- and red-ringed snake, *Rhyncelaps bertholdi*, which has a bad reputation amongst them, from the Kimberleys in the north to the Nullarbor Plains in the south".

In *The Argus* of January 5, 1927, is an article on dangerous lizards. The writer of this states that the commonest lizards capable of inflicting an injurious bite are the frilled or Jew lizards (*Amphibolurus*), the blue-tongued and stump-tailed skinks (*Tiliqua* and *Trachysaurus*) and the monitor or goanna (*Varanus*). It is added that recently in New South Wales the bite of a frilled lizard ended fatally, and a number of cases of death following the bite of a blue-tongued lizard had been recorded. I am quite unaware of any of these cases having been properly investigated. If sepsis has followed wounds inflicted by lizards, such might cause death, but this is more an accidental association than a necessary accompaniment of the bite.

Injuries from Fishes.

Injury from a Groper.—G. P. Whitley, of the Australian Museum, gives the following information about this fish:

In his well known book, "The Confessions of a Beachcomber", the late E. J. Banfield wrote that the groper will follow a man in the water with dogged determination foreign to the nervous, suspicious shark. Recently a vigorous young black boy was attacked by a groper while diving for *bêche-de-mer*. The fish took the boy's head into its capacious mouth, mauling him severely about the head and shoulders, and but for his valiant and determined struggles would doubtless have succeeded in killing him.

The late Allan R. McCulloch once told me that divers are afraid to descend to the treasures of the wrecked Spanish galleons which lie around Boot Reef in the northern Coral Sea because of the large gropers there. Whether they belong to the same species as the Queensland groper or not, nobody has been courageous enough to determine.

Injuries from Sharks.—An account of Australian cases, since the last issue of this series, has been recently contributed by me[11] to *The Journal of Tropical Medicine and Hygiene*.

Injuries from Stingrays.—A friend of mine in Western Australia, an elderly man, was wounded in the left leg, near the ankle, by a stingray.

He felt no pain, but numbness, drops of perspiration came out before he was able to reach home and the leg felt light, the symptoms all suggesting some absorption by the nerves.

Dr. John Burgess,[12] of Cowes, Phillip Island, Victoria, describes in a letter three cases of injuries from stingrays.

The patients experienced intense pain, in one case agonizing and in another giving rise to a condition of shock with ashen colour and general weakness. The pain disappeared in from half to one hour. In one case where there was agonizing pain, the stingray was caught and was only about the size of the palm of the hand.

Dr. Paul G. Dane[13] recorded a case from Brighton, Victoria.

There was a small bayonet-shaped puncture on the dorsum of the foot, the patient suffering great pain, which was complained of as extending up to the knee. The face was pale and the pulse rate over 100. Incisions were made and the foot placed in a bucket of hot hypertonic salt solution and a rubber bandage placed round the thigh. In about an hour the pain was relieved.

In January, 1923, at Port Victoria, South Australia, a man, while trying to spear buttterfish, trod on a stingray; the teeth penetrated the tennis shoe he was wearing and entered the foot, causing severe pain.

Injury from the Spike of the Bugler Fish (Long-Snouted Boar-Fish, Pentaceropsis recurvirostris).—The bugler fish contains a sharp spike on the end of the nose.

One weighing about twenty pounds was hauled into a boat off Beauty Point, near Launceston, Tasmania, and as it was released from the net, sprang into the air and came down head first, piercing the patient's instep. He was taken to the public hospital at Launceston, and I am indebted to Dr. E. W. Buland, the Medical Superintendent, for an account of his condition.

Two inches of the spike were supposed to be still in position in the interphalangeal space, between the big and second toes on the plantar aspect. The foot presented all the appearances of an irritating foreign body under the skin, but when the tissues were thoroughly opened up, no sign of any foreign body could be found. Presumably the appearances were due to some toxic substance introduced by the spike.

Injuries from the Stone-Fish or Dornon (Synanceja horrida).—The late A. R. McCulloch[14] describes the stone fish or Dornon and gives a photograph of it. It is met with freely in the Torres Straits. He states that if kicked against unawares, or if the upstanding spines are trodden upon, the patient suffers intense pain or may even die.

In 1928 J. V. Duhig and Gwen Jones[15][16] published accounts of investigational work on the venom of *Synanceja horrida* in which a hæmotoxin was found. These papers should be consulted.

G. P. Whitley and W. Boardman[17] describe the following case of injury from the spine of the stone-fish:

We met a fisherman (at Low Isles, Great Barrier Reef) who had had his finger scratched by a stone-fish spine. He suffered intense agony for fifteen hours, and his finger became so stiff that he could not bend it, the poison seeming to affect the nerves rather than the blood. He felt at times as if someone were twisting each of his vertebræ, one after the other, right down the spine, and was unable to obtain relief from doctors or herbalists. At times the pain was so intense that only the thought of his wife and family restrained him from committing suicide. Eventually he recovered, but cases of death from stone-fish poisoning are by no means uncommon.

Toxic Symptoms Attributed to Eating Sweep (Scorpis æquipinnis Rich.).—Dr. H. Carew Nott has given me the following details of symptoms of poisoning shown by five persons out of a party of nine at Kangaroo Island on December 29, 1928.

It was noticed that one of the fish had an unusually thick skin with few scales, and that the flesh was reddish. Symptoms in one case commenced during the meal, and in others up till a couple of hours afterwards. It would seem as though some irritant poison was present, probably in the skin of the fish. The details as supplied are the following:

About 1 or 2 o'clock in the afternoon nine sweep were caught at Pennington Bay, K.I. All these fish, according to several experienced amateur fishermen, were undoubtedly sweep. Two of these were large, about two and a half and two and a quarter pounds respectively, and Mr. W. Gunson noticed on cleaning them that one had unusually thick skin with comparatively few scales; the cook subsequently remarked on this also and, too, that the flesh was reddish in colour. These two fish were cut up, fried in batter and eaten about 8.30 o'clock on the same evening. Subsequently five of the party had symptoms of poisoning identical in each case—severe epigastric pain, feeling of chilliness without apparent rise in temperature, sweating or shivering, followed by excessive vomiting and relief; there was no diarrhœa and all were practically well the next day. The onset of these symptoms varied from during eating the fish (in this case nothing further was eaten and the individual vomited about thirty times during the evening) to a couple of hours after the meal. Both fish were cooked in the same batter, and one of the sufferers, a child of nine years, had mostly skin, being fond of this, and only a tiny amount of flesh for her portion. The four persons who escaped had not the slightest symptoms of poisoning. The remaining seven smaller sweep were cooked the same way and eaten for breakfast next morning without any ill effects. There had been nothing else drunk or eaten during the day by the five sufferers that had not been shared by one or more of those not affected.

Dr. Nott made the following comment:

By a process of exclusion it seems to me practically certain that the origin of the trouble was in one of the large sweep, probably in the skin.

Injuries from Crustaceans.

Cirolana Woodjonesi Hale.—Dr. J. W. Yeatman has called my attention to the irritation received by persons bathing about sunset along the Gulf of St. Vincent, caused by small crab-like creatures

about half an inch long, of a grey colour, which proved on identification by the South Australian Museum authorities to be the above species. The creatures are hard to see in the water; they readily attack bait. Dr. Yeatman's experience of their effects was that he had a feeling of sharp little pricks whilst in the water, and later discovered a scattered, punctiform, bright red rash, mostly about the body and thighs. In some persons urticarial wheals were also present. The rash lasted about thirty-six hours and was just painful enough to be annoying. Methylated spirits is the popular remedy. The South Australian Museum authorities state that this species is free-swimming, a scavenger, and that it will attack living fishes. It causes irritation to bathers by biting small pieces of skin with its sharp-edged tridentate mandibles and tearing at the tiny wound with the first maxillæ, which are armed with strong spines. Four species of the genus occur in South Australia and fourteen species in Australia as a whole. They belong to the family *Eurydicidæ*, one of the Cymothoid families of Isopoda.

In *The Register* of January 12, 1928, a short reference is made to sea-lice and shell-fish swarming at that time in the sea at the Adelaide beaches during the heat wave.

Persons bathing felt an itching and burning sensation on the arms and legs, so that more bathers were on the beach than in the water. A moonlight night and a calm sea specially attracted the smooth sea louse into the shallows.

Injuries from Arachnida.

Mites.

A Skin Rash Probably Due to Mites.—In an unsigned article in the *Agricultural Gazette of New South Wales*[18] an account is given of men suffering severely on handling and chaffing wheat in April and May, 1921, at the Grafton Experimental Farm.

There was much irritation and skin rashes which spread over the body, forming white blisters. Mr. W. W. Froggatt suggested that the condition was probably due to harvest mites, but none were found in the sample of grain submitted, having probably fallen off in transit, though there was present a very dark-coloured grain weevil and a small ant.

Tyroglyphus sp.—In an unsigned article in *Health*[19] a description is given of skin irritation, due to mites identified as *Tyroglyphus* sp.

The irritation occurred in a number of workmen engaged in upholstering work on motor cars in Brisbane. The irritation resembled scabies. There were no constitutional disturbances or absence from duty. It was thought that infestation probably occurred in Italy or on the voyage.

Liponyssus bagoti.—F. G. Holdaway[20] gives a "Note on the Occurrence of the Rat Mite, *Liponyssus bagoti*, in South Australia, together with Descriptions of Certain Stages".

These mites had been found giving rise to an itching irritation in certain business places in Adelaide, as they have given rise similarly to irritation in other parts of Australia.

Harvest Mites.—Harvest mites have been reported as causing irritation of the skin.

After the Rendelsham railway smash between Millicent and Beachport, early in January, 1928, a number of the passengers were gathered together near a thick growth of tea-tree, a spot infested with what was locally called the "red spider", which produces the tea-tree itch. Intolerable itching was the result.

This species of harvest or larval Trombidiid mite has recently been investigated by the late Mr. Stanley Hirst,[21] who has prepared a description of it and has identified it as *Trombicula hirsti* Sambon. This species is the "scrub itch mite" of Northern Queensland, from which locality L. Sambon described it in 1927. The type came from Innisfail, and Hirst has identified specimens also from Tully. In South Australia the mites extend from Kingston to Robe and are found also near Mount Gambier. Hirst says the species is probably identical with *Trombicula pseudo-akamushi*, described by the Japanese Hatori. It occurs also in Sumatra and the Malay Peninsula, as well as in Japan and Australia. This species so far is not known to convey any specific disease, but Hirst points out that *Trombicula akamushi* Brumpt and *Trombicula deliensis* Walch are known to transmit one of the forms of tropical pseudo-typhus.

Trombicula (*Leeuwenhoekia*) *australiensis.*—The late Stanley Hirst[22] has described a new harvest bug (*Leeuwenhoekia australiensis*) attacking human beings in Sydney, New South Wales.

The specimens had been sent to him by the late Dr. E. W. Ferguson. Dr. Ferguson's accompanying letter stated that the mites attacked people working in the garden and caused great irritation. The attacks recurred annually. The mites were apparently present from about October onwards till April. They seem to prefer the moister surfaces, such as the axilla, under corsets and waist-bands, round the eyelids *et cetera*. The mites apparently actually burrowed into the skin. Ashfield was the suburb of Sydney where the cases chiefly occurred.

The mites belong to the family *Trombidiidæ*, but the adults are unknown. Hirst states that this is the first recorded instance of a mite of the genus *Leeuwenhoekia* attacking human beings. The species is known also from Sumatra.

Sarcoptes Mites.—In a discussion on scabies before the Sections of Dermatology and Comparative Medicine of the Royal Society of Medicine, held on February 19, 1931, A. W. Noel Pillers[23] refers to a human infection with *Sarcoptes* from a wombat having been recorded. As the wombat is a purely Australian animal, this reference has interest for us, even though the infection may have been contracted from an animal in a zoological gardens outside Australia. I have not been able to ascertain the source of Mr. Noel Pillers's information.

Demodex.

Dr. Hermann Lawrence[25] has described a skin disease occurring in adults in Australia which resembles *impetigo contagiosa*, though differing from this in the greater tendency to form rings and in the border of the skin being more definitely raised. Pustulation also was not so severe, and the lesions did not respond so rapidly to the usual simple antiparasitic remedies employed. In some cases the patients have attributed the eruption to shaving, and it may be found in other regions, such

as the forehead, neck and nose. In the lesions are found an extraordinary number of *Demodex folliculorum*. Dr. Lawrence is inclined to think that these mites play some part, at least, in the ætiology of the condition. He refers to an odd specimen of demodex being often found in sebaceous matter squeezed from the nose. In this disease the demodex are very much more numerous. He points out that in veterinary practice the demodex is considered pathogenic, giving rise to one form of mange. I have myself seen large numbers of demodex in a follicular skin eruption in pigs, and have also found the bovine form present in sebaceous plugs in the skin of a bull.

Dr. Lawrence[25] contributed a further note to the Australasian Medical Congress in Brisbane in 1920. He had seen several further cases in men who thought that they had contracted the disease at the barber's. It differs from sycosis in that the hair follicles are not particularly affected and that the circinate serous oozing spots may occur upon the forehead and neck as well as upon the hairy parts of the face. The late Dr. Harrold had seen similar cases in Adelaide.

Dr. Lawrence[26] has frequently found demodex in the scrapings from keratoses of patients with kerotic skins.

Dr. Hermann Lawrence and Dr. Robert Brodie[17] have recently called attention to a skin disease for which Ayres had coined the term *"Pityriasis folliculorum demodex"*. Since reading his article they had met with four undoubted cases in Australia. The patients had rough, scaly patches with discoloration. Large numbers of the parasites were present. Whilst having seen four cases of the pityriasis in the five months they had only seen one case of a demodex impetigo during the same time.

Ticks.

A discussion on tick bites, together with a review of recent cases, has been published by Dr. I. M. Mackerras.[28] In this article the cases recorded and their symptomatology are epitomized. Included are references to the late Dr. E. W. Ferguson's paper[29] on "Deaths from Tick Paralysis in Human Beings", and to Dr. H. St. Leger Moss's article, "A Case of Tick Paralysis".[30] The tick in the latter case was identified as *Ixodes holocyclus* by Dr. E. W. Ferguson.[31] Dr. W. Stewart McKay[32] has given an account of the anatomy and poison of the dog tick (*Ixodes holocyclus*), based on experience with his own dog. Recovery occurs under suitable treatment.

Dr. C. W. Sinclair,[33] of Longueville, Sydney, has recently described a fatal case of tick bite.

The patient was a delicate girl of three and a half years. The tick probably attached itself on November 16, 1929, when indefinite irritability and malaise began. On November 19 the pupils were dilated; on November 20 the gait was slightly unsteady and the tick was found on the scalp on the left parietal bone with considerable swelling around it. Next morning the girl's legs were paralysed and there was paralysis of the *sphincter ani*. During the day paralysis of deglutition developed and she died early on November 22. The temperature rose to 38·9° C. (102° F.) before death, when the pulse, previously strong and normal, became rapid and feeble.

Poisonous Spiders.

For many years the red-backed spider (*Latrodectus hasseltii* Thorell) has been known as causing from time to time serious injury to man. It is only, however, within the last few years that we have become aware in Australia that spiders belonging to other genera may bite man with severe and even fatal results. The species concerned are two trap-door spiders, *Atrax robustus* and *Atrax formidabilis*.

Bites from the Trap-Door Spiders (*Atrax robustus* O. P. Cambr. and *Atrax formidabilis* Rainbow).—Anthony Musgrave,[34][35] Entomologist of the Australian Museum, has given two accounts of injuries caused by these trap-door spiders.

Atrax robustus gave rise to the death of a baby boy at Thornleigh, near Sydney, in February, 1927. The little boy had been sitting on some laundry steps at his parents' home when he suddenly jumped up with an agonizing scream. There was a spot of blood on the little finger. When seen by a doctor the child was unconscious, deeply cyanosed and in a state of convulsive spasm. Death resulted. The spider was identified as *Euctimena tibialis* Rainbow, which turns out to be the male of *Atrax robustus*, the male of which species had previously been unknown.

In a second case of a bite from this spider, the patient was a male, aged forty-two, who was bitten on the foot at six o'clock in the morning. He felt a severe prick, and two small punctures were visible. In thirty-five minutes there was a peculiar numbness of the tongue and loss of taste, with a twinging pain in the tongue and lips similar to that caused by an electric current. In one and a half hours the pain had extended to both hands and feet, with a strange sensation as though something cold was touching the skin. In two hours pain was present in both arms and legs, and there was excessive salivation with a discharge of mucus from the nose. There was twitching of the facial muscles, especially those of the lips. Four hours after the bite the patient felt considerable depression, the eyes were watery and the eyelids could be controlled with difficulty. There was nausea and a choking feeling in the throat and a sensation of obstruction in the nose. In four and a half hours the vision became very blurred, the images overlapping, and there was difficulty in controlling the limbs, the hands and feet all being numbed and extremely sensitive to cold. In five and a half hours the symptoms were more severe. The patient found it impossible to use a typewriter. In seven and a half hours the feeling of tingling had extended to the hips, shoulders and back. All the symptoms continued until late at night, the bite having occurred at six in the morning. Next day there was considerable improvement, though the tingling was still present. He felt tired, depressed and numb. On the second day only the hands and feet tingled at intervals and the bitten foot was slightly sore.

R. N. Beazley[36] has recorded the death of a married woman of forty-six at Wahroonga, near Sydney, on January 8, 1930, from being bitten on the thumb by a trap-door spider, later identified as *Atrax robustus*.

The patient, when seen forty minutes after being bitten, was pale, cold and clammy, except for the red, congested face, had a thready and almost uncountable pulse and a systolic blood pressure of fifty-five millimetres of mercury. Incessant retching was followed after an hour by increasing dyspnœa with signs of pulmonary œdema and frequent paroxysms of laryngeal spasm. There were as yet no other muscular spasms or tremors, the mind seemed clear, and there was no complaint of pain and no redness or

swelling of the thumb. Four hours after being bitten the patient was restless but conscious, with stertorous breathing and moderate cyanosis. Every ten to twenty minutes there were clonic contractions of the arms and legs with laryngeal inspiratory stridor. There was slight spasticity of the limbs between the spasms. The pupils were small and did not react to light. At the site of injury were two very minute punctures with, on excision, two small thrombosed vessels in their neighbourhood. The patient was comatose six hours after the bite, but still had laryngeal spasms. Death occurred suddenly three hours later. No tourniquet had been applied just after the bite; excision of the injured area took place about four hours later, when potassium permanganate was applied. The *post mortem* examination revealed no lesions of special importance.

The Bite of the Red-Backed Spider, Latrodectus hasseltii.—Dr. C. H. Kellaway[37][45] has carried out a series of experiments on, and given a full account of, the venoms of our red-backed spider. The symptoms following its bite agree with those recorded of the bites of other species of *Latrodectus* from foreign parts. The symptoms in animals following a natural bite suggest that the venom has a neurotoxic action. In guinea-pigs bronchial constriction plays a prominent rôle in causing death.

In 1925, in *Health*,[38] appeared an unsigned note on the symptoms following the bite of a red-backed spider.

Dr. E. H. Miles,[39] of Yeoval, New South Wales, sums up the symptoms in nine cases.

The bites reported by him were usually about the genitals, from the red-backed spider. None were fatal. Symptoms appeared generally after two to three hours. There were intense burning pain at the site of the lesion persisting for some days, a feeling of numbness, mostly in the feet and legs, violent shivering and profuse sweating. Scarification and rubbing in a solution of ammonia seemed to abate the more severe general symptoms in two cases.

Dr. E. S. Jackson[40] gives details of a fatal case in a small boy of three or four years that occurred many years ago.

The child had been bitten in the groin apparently twice by a red-backed spider, which was found and killed, eighteen hours before admission to the Brisbane Hospital. There were two punctures half an inch apart. The boy was dull and heavy, but, as far as he remembered, not complaining of any great distress. Death ensued thirty-six hours after admission. *Post mortem* examination revealed no lesions other than enlargement and intense engorgement of the chain of glands lying alongside the spinal column up as far as the diaphragm.

Dr. Stewart McKay,[41] in a letter, discusses in general red-backed spider bite, tick bite, scorpion stings and snake bite, but does not give any Australian examples.

Dr. H. V. Lethbridge[42] notes that he had treated about thirty cases of bites from the "jockey spider" or red-backed spider. Of these all had been extremely ill, three of them dangerously, and one, a boy of eighteen years, seemed hopeless. He knew of three deaths from this cause.

Dr. Arthur Watkins[43] states that his patients, all males, had been bitten on the *glans penis*, scrotum, groins or some part covered by the clothes. He notes that a small black spider of about the same size as *Latrodectus hasseltii* gave only a severe local reaction, the part being infiltrated and hard. He had found ichthyol useful.

Dr. Hales,[44] of Brewarrina, has recorded a bite from *Latrodectus hasseltii*.

The patient was a girl, aged five years; she was bitten on the hand. A ligature was applied fifteen minutes later. Sixteen hours afterwards the hand and face were swollen, the temperature was slightly raised and she was sweating freely. She continued to sweat freely for nine days. At the end of fifty hours she lost her power of speech and swallowing for a few minutes. The face was swollen and the temperature was 38·4° C. (101·2° F.). On the fifth day an irritable rash developed over the body.

Dr. Lethbridge, Dr. Holmes à Court and Dr. Hales[45] referred to cases, some included in those mentioned above, at the Australasian Medical Congress in Sydney in 1929.

Dr. Eric Broadbent, of Bordertown, South Australia, has given me notes on a case which occurred at the beginning of 1930.

The patient was out sewing bags of wheat and whilst taking a bag off a heap of new ones he was bitten by a red-backed spider. The forearm where he was bitten, soon became swollen and red and he came in and consulted Dr. Broadbent. When seen, he had a red, swollen area and in the middle there was a large bleb filled with a dark serous fluid. He looked ill, complained of feeling sick, and had a headache and throbbing pains at the site of the trouble. The bleb was opened and hot biniodide of mercury foments were used every hour. Next day the trouble had spread a little. The same treatment was continued. At this stage he had diarrhœa and the tongue was very heavily furred. The third day he seemed better and a large slough about the size of a two-shilling piece started to come away. After this he had an uneventful recovery and was sent home on the tenth day.

The peculiarity of the case was the persistent headache, which would not yield to the usual drugs. Red-backed spiders are plentiful in the neighbourhood of Bordertown.

In February, 1929, appeared a newspaper reference to a patient, Mrs. G.W., who had been bitten on the finger by a red-backed spider.

On inquiry, Dr. G. M. Whish, of Young, said that it was very doubtful whether the patient had been bitten by a spider. She had a septic sore on the finger, causing some slight general infection, with suppuration round the left shoulder joint and cellulitis of the left calf.

Scorpions.

Alfred Giles[46] has described his personal experience of scorpion bites received in February, 1871, in Central Australia, near the junction of the Hugh with the Finke.

The scorpions . . . were of an enormous size, and I was stung twice within fifteen minutes . . . on my arm and back of the neck. The sting is very painful, like a coal of fire, and lasts for about twenty minutes, but fortunately they were not large ones.

A Chelifer Caught on a Patient with Acariasis.—Chelifers are very small scorpion-like arachnids that prey on minuter forms of life. The following reference, from William Macleay's diary,[47] to specimens received is of interest, partly because the specimen was presented to Macleay in 1874 by Dr. James Cox, for many years Lecturer in Medicine in the University of Sydney and a famous conchologist.

A chelifer got at the same house as one he gave me on a former occasion, taken off a woman suffering from acariasis. No doubt the chelifer was feeding on the acari.

If the patient was suffering from scabies, it is difficult to understand how the chelifer was able to secure its prey.

Injuries Due to Myriapods.

Centipedes.

Keith McKeown,[48] in an article on Australian centipedes and their bites, mentions several cases that came under his own observation, in which the bite was accompanied by severe nerve pains and, on recovery, the whole of the inflamed area surrounding the punctures gradually sloughed out like the core of a boil—features apparently typical of recorded cases. Two common species appear to be poisonous, the distinguishing features of which he gives as follows:

Ethmostigmus rubripes is the largest of the common species found in Australia; it has also a wide distribution in other countries. It measures up to about eight inches in length, and may be distinguished by its cusped teeth, the teeth being three in number; the stigmata, or openings along the sides of the body are uncovered, and situated on the second and every alternate segment following to the end of the body.

Scolopendra morsitans is rather smaller than the previous species, and has four to five teeth on each side; the stigmata are closed by means of valves. This centipede is cosmopolitan in its range. In coloration these centipedes are generally similar, and are of shades of brown, green and yellow; they vary considerably, however, so that colour does not prove a safe guide for identification.

A specimen of *Ethomostigmus rubripes*, six inches in length, was apparently responsible for killing a dog at Drummoyne in November, 1929. McKeown states that:

Although the long curved fangs are the weapons of aggression, the terminal claws are more or less poisonous and leave a track of inflammation where they are allowed to come in contact with the naked skin.

Millipedes.

In 1929 Dr. Helen Mayo, of Adelaide, submitted a specimen, identified by Professor Harvey Johnston as a millipede, *Julus* sp., which was supposed to have been passed by a child. It is doubtful whether this was the case. If it were, the creature had evidently been accidentally ingested, merely passing through the intestine without taking up a temporary abode therein.

Injuries from Mollusca.

The Giant Clam.—G. P. Whitley and W. Boardman[49] state that the giant clam (*Tridacna gigas*), which occurs on the Outer Barrier Reef and is of very great size, is much dreaded by divers, for if touched by leg or arm when open the shells close and no amount of human force can separate them, the victim being held in a vice-like grip till he drowns.

Cephalopoda.—A note in the *Australian Museum Magazine*[50] mentions a bite which drew blood, at Port Hacking, from the long parrot-like beak of a small cuttlefish, only five to seven inches long.

Injuries from Annulata.

Intractable Bleeding from a Leech Bite.—Dr. S. J. Woolnough,[51] of Blackheath, has described the case of a child of three and a half years, brought to him on Christmas Day, 1927.

The vagina was full of blood and blood had soaked through a large bath towel and well into an eiderdown quilt during a twenty-mile drive. A leech was found near by full of blood. The vagina, from which the bleeding appeared to have come, was packed with weak adrenalin and further bleeding soon stopped.

Another child an hour or so later had been bitten by a leech on the leg.

Marine Worms.—A. Musgrave[52] states that at the Capricorn Group, off the Queensland Coast, marine worms armed with tiny siliceous spicules were encountered in breaking off coral fragments, their poisonous spicules entering the skin and setting up a severe irritation.

Injuries from Echinoderms.

Sea Urchins.—The foot-long spines of the needle-spined urchin (*Centrechinus*), according to G. P. Whitley and W. Boardman,[49] are apt to pierce the tissues, and owing to the liability of the brittle points to break off, poisonous effects are apt to result.

Injuries from Cœlenterates.

Trachymedusæ.—Dr. A. E. Stenning,[53] of Onslow, Western Australia, describes a case of severe poisoning by a jelly fish.

The patient was a man, aged twenty-seven, who was swimming in the sea at Onslow when he felt a sting on the shoulder and noticed web-like material (the tentacles of jelly-fish) over this area. Pain and numbness immediately developed in the shoulder. Slight frothing at the mouth occurred and intense vomiting within five minutes. The pain soon became generalized, with cramps in the abdomen. The pain was specially severe across the loins, in the back of the legs and across the shoulders. On examination a wheal was noticed on the top of the right shoulder which, with adjacent parts, was considerably swollen and cyanotic. The patient was extremely collapsed and was sweating profusely and was evidently in extreme pain. The eyes were suffused and the pupils dilated. There was definite tremor, the extremities were cold and numb, the knee reflexes were exaggerated. Morphine and stimulants were given and the patient left the hospital in three days, though having lost 4·5 kilograms (ten pounds) in weight.

Twelve months previously, apparently about March, 1927, half a dozen similar cases had occurred, presumably due to stings of these animals.

The Stings of Sea Anemones.—Dr. A. A. Livingstone[54] states that *Actinodendron plumosum*, a graceful Great Barrier Reef sea anemone, possesses powerful stinging properties which have earned for it the reputation of being one of the most unpleasant sea anemones to handle. One authority states that the sting is almost as severe as that of a nettle and that the resultant rash may remain visible for nearly a week.

Protozoa.

Trichomonas Vaginitis.—H. O. Lethbridge,[55] of Narandera, has called attention to the not infrequent occurrence of *Trichomonas vaginalis* in the vagina giving rise to considerable irritation, a rather profuse yellowish discharge, sleepless nights and an unpleasant odour. Microscopically, gono-

cocci are absent unless there happen to be two separate infections. Dr. Lethbridge had had three cases in which live trichomonads had been present.

H. G. Furnell[56] records two further cases associated with vulvo-vaginal discharges. In one pneumococci were also present and the vagina was intensely red and swollen. In the other gonococci were detected.

Urinary Infection with Trichomonas Vaginalis in the Male.—D. B. Rosenthal[57] describes a case of urinary infection with *Trichomonas vaginalis* in a male.

The patient was a widower, aged seventy-two years, symptoms commencing five weeks after laparotomy for cholelithiasis. Dr. Rosenthal considered that the pyelitis and cystitis were due to a coliform infection and that the trichomonads were coincidental. On making the urine alkaline, they disappeared and did not reappear when later it became acid.

A subleader in the same issue of this journal[58] briefly discusses these Australian cases and other recent literature, and calls attention to the doubt that exists as to whether these organisms are really pathogenic.

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After the publication of the last number of this series, Dr. Kevin Byrne,[59] of Lakemba, New South Wales, published notes on the following as causing injury to man: Crocodiles, turtles, sea-snakes, flying fish, and the stone-fish (*Synanceja horrida*), and an article on coral cut. The late Lieutenant W. J. Paradice[60] also contributed an interesting article containing accounts of bites of eels, pricks from the gill rakers of fish, wounds from the spines of the piked dogfish, stings by the spines of catfish and by the spine of a stingray, Chinaman fish poisoning, poisoning by the flesh of toados, injuries from larval ticks, the irritation of polychæta worms, stings by the spines of sea urchins, coral cuts and jelly fish slime.

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Reports of Cases.

SOME NOTES ON A CASE OF ARSENICAL POLYNEURITIS.

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N.S.T., A MALE, single, aged nineteen, a clerk by occupation, was seen by me in consultation with Dr. J. B. F. MacKenzie at the Newcastle General Hospital. The patient complained of pains in the lower extremities, with numbness and tingling, weakness of hands and feet, and inability to walk. The onset of his illness was given as seven weeks ago, when he was stated to have suffered from an attack of vomiting and diarrhœa with the daily passage of numerous watery stools which necessitated his remaining in bed for a period of two weeks. When the patient got up he noticed weakness of the wrist, elbow and knee joints, but no pain in the joints; the soles of the feet were painful when walking; the pain gradually became more persistent and intense up till his admission to hospital, when it was continuous and more severe at night time. He was unable to balance himself or walk without the aid of some support or carry heavy objects. He noticed that both his hands and his feet were inclined to drop. There was some hoarseness of his voice six weeks prior to admission. There was no vertigo and no headache, and the sight was unimpaired. The family history was irrelevant. He had had no previous illnesses of note, he denied venereal infection and excessive use of tobacco. He gave a history of moderate potus, and admitted he had indulged in excessive sexual intercourse nightly for the previous five months up to the onset of his illness. He had been working as a clerk in a garage next to a paint shop where "Duco" and paint spraying was carried out, but none of his fellow employees had suffered from any similar malady. His water supply and dietary had remained unaltered for some time.

On examination the patient appeared obviously ill and irritable on account of pain and discomfort he was suffering. Muscular weakness was apparent in the upper and lower extremities, and a bilateral wrist and foot drop were present and an ataxic gait. Temperature, pulse and respiration were within normal limits. There was hypotonia and wasting of the muscles of the calves and forearms, and excessive wasting of the intrinsic muscles of the hands (see accompanying illustration). There was excessive tenderness of the muscles to touch and pressure, and he complained of pain on passive movements of the extremities. The abdominal and deep reflexes were absent. The plantar reflexes were flexor. There was impairment of tactile and thermal sensibility over the peripheral portions of both extremities; this was more marked in the lower, with scattered areas of hyperæsthesia. The stereognostic sense was impaired. The pupils were somewhat dilated, but reacted readily to light and accommodation. The cranial nerves and sphincters were functioning normally. The other systems appeared normal and there was absence of any pigmentation of the skin. An interesting observation was the discovery of transverse white bands on the nails of the fingers and toes. A diagnosis of arsenical polyneuritis was made, together with a recommendation that clippings of the patient's hair be submitted to an analyst for examination for the presence of arsenic. Intravenous medication with sodium thiosulphate was recommended. The blood serum gave no reaction to

Figure showing hands of patient with arsenical polyneuritis and transverse white bands on the nails.

the Wassermann and Kline tests. Lumbar puncture revealed a clear cerebro-spinal fluid under increased pressure; it did not react to the Wassermann test. Felix mass was given and the stools searched for ova without result. A test meal revealed free hydrochloric acid in normal amount. The blood picture was as follows:

Red blood cells, per cubic millimetre ..	4,540,000
Hæmoglobin value	90%
Colour index	0·99
Leucocytes, per cubic millimetre	3,700
Neutrophile cells	41%
Small lymphocytes	40%
Large lymphocytes	8%
Large mononuclear cells	6%
Eosinophile cells	5%

Slight variation in size and shape of red cells was noted. A few cells showed polychromasia and stippling. No nucleated red cells were seen and no reticulocytes. The blood platelets were normal.

The analyst's report on 2·85 grammes of the patient's hair from the head revealed the presence of arsenic in the proportion of 4·2 milligrammes per 100 grammes.

A history of heavy potus (beer) prior to the onset of the illness was subsequently obtained from the patient, but this factor as a source of the arsenic might be excluded in view of the methods of local manufacture and provisions of the *Pure Foods Acts*.

Comment.

The diagnosis in this case was at first doubtful owing to the rapid development of the paresis and muscular atrophies, but the normal temperature, pulse and respiration ratios, together with the sensory anomalies, suggested a polyneuritis. The gastro-intestinal attack as a prodrome of the malady is also worthy of note. The patient, as is usual in such cases, was entirely unaware of the cause of his malady, and it was necessary to attempt to elucidate the ætiological factor. The transverse bands present on the nails of the fingers and toes have been regarded as actual

deposits of arsenic,[1] and it was on their presence alone that a diagnosis of arsenic as the ætiological factor was made before this was confirmed by a chemical examination of the hair.

Hassin[2] has commented on the scarcity of contributions on this subject to date, and it is with a view to assisting in estimating the relative value of the white bands in the nails and the presence or absence of arsenic in appreciable quantities in the hair that these notes have been recorded.

Acknowledgement.

My thanks are due to Dr. J. B. F. MacKenzie for permission to publish clinical notes in connexion with the case.

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Reviews.

A GUIDE FOR DIABETICS.

"TEN years ago diabetes spelt death. Today, since the advent of insulin, *the diabetic's worst enemies are his own recklessness, and old age.* Once there was no hope, but today we may appreciate the words of that great American, Dr. Joslin: 'Diabetes is a good disease with bad companions.' The bad companions are always matters for your doctor, *but the diabetes is always a matter for yourself.*" These are the concluding remarks of the opening chapter of "Notes for Diabetics", an excellent little book by "One of Them".[1] Diabetes is the one disease above all others of which the patient must have more than superficial knowledge and in the treatment of which he must cooperate. Given understanding and cooperation, it is possible for a diabetic to live a comfortable and useful life. He must be continually on the watch, he must be able to recognize the danger signals and he must know how to meet them. Many books have been written for the use of diabetic patients, and this, the latest, by an Australian patient who is not a medical graduate, is a welcome addition to the number.

The author of this book (his identity is not disclosed) is obviously a well informed man with a sense of humour and a *joie de vivre*. His advice is sound—he has learned his lesson well—and the book may be recommended to medical practitioners with every confidence. Professor Harvey Sutton, of the University of Sydney, in a foreword writes: "This book is . . . of the greatest possible value to every diabetic. It is quite remarkable in its vivid description of disease experience and in its accurate outline of the methods to be followed, under his doctor's orders, by the patient, in the hypodermic use of insulin and in urine testing. . . . Here is a book, both inexpensive and thoroughly intelligible, in which the dry bones of knowledge are clothed with lively wit and practical wisdom illuminated by flashes of real genius."

After an opening chapter of general remarks comes one on diabetic knowledge. In this chapter the author points out that unless the diabetic is equipped with something more than ordinary knowledge of his complaint, he will never be safe out of his doctor's sight. Then follow chapters on insulin and insulin reaction and on how to take insulin. The chapter on tests for sugar in the urine, diacetic acid, acetone and glucose tolerance is short and to the point. In discussing quack remedies, the author gives the sound advice that diabetics should stick to the diet and insulin prescribed by the medical attendant and let others experiment on themselves as they feel inclined. Then follow several chapters on diet, on carbohydrates, proteins and fats, and on how to make the best of diet. The chapter on coma and insulin reaction is one of the best in the book. The author has experienced both conditions and gives convincing advice; he has drawn up a table in which the symptoms of the two conditions are contrasted side by side. Under the heading "Exercise and General Hygiene" considerable space is rightly given to methods of caring for the feet: "Learn to 'manicure' the feet first and the hands afterwards."

In conclusion we can do no better than quote the words of Professor Sutton that this is a book which "every medical practitioner should possess and recommend to his diabetic patients".

[1] "Notes for Diabetics", by One of Them, with foreword by Harvey Sutton, M.B.; 1931. Australia: Angus and Robertson, Limited. Crown 8vo., pp. 98, with illustrations.

THE LITERATURE OF THE VERTEBRATES.

THE Oxford University Press can always be counted on for publication that is above the average in style, and C. A. Wood's "Introduction to the Literature of Vertebrate Zoology" is no exception, for the print, paper and general lay-out are most attractive and beyond reproach.[1] The cost of production must have been very considerable, the volume consisting of 643 quarto pages.

The abbreviated title does not, however, provide a complete clue to the contents. The title page adds that the work is "based chiefly on the titles in the Blacker Library of Zoology, the Emma Shearer Wood Library of Ornithology, the Bibliotheca Osleriana and other libraries of the McGill University, Montreal". The preface further sets out that the volume is a six years' effort intended to assist students of vertebrate zoology by indexing the literature of the libraries named. One gathers here that the publication itself has been financed by those libraries.

It will be recognized at once that any guide to literature prepared in this way is bound to suffer from certain limitations. This might have been the slightest of criticisms, for it is clear at the outset that the McGill University is remarkably well supplied with zoological literature and literature of use to students of medicine. The list of titles collected since 1920, when the Blacker Library was founded, makes us gasp. Would that someone would endow the science side of any Australian library in the same munificent manner! We have nothing like it.

Unfortunately, we cannot feel that the contents of the volume are up to the standard of the publishers, or that the compilers have done anything like the best with the material at their disposal. No doubt the volume is of value to anyone looking up vertebrate literature, more especially perhaps to systematists and in particular to ornithologists. But the serious worker along special lines will soon have to turn to the well-known works of reference like the *Zoological Record et cetera*. It is amazing how scrappily and carelessly arranged is the first section, which is supposed to review the literature of vertebrate zoology. Thus, on a page headed "Treatises on Anthropology" we read:

> Somewhat later there was published a work of classic importance, W. P. Pycraft's "Courtship of Animals", 1913. S. J. Holmes, "Studies in Animal Behaviour", published in 1916, is well worth the student's attention. Still later appeared a text book by T. H. Morgan and others, "The Mechanism of Mendelian Heredity", 1919.

It is difficult to find any criterion that would justify bringing these three works together, but worse follows. Five lines below the above appears the statement: "E. N. Harvey published in 1920 'The Nature of Animal Life'." Now the real title of Harvey's work is "The Nature of Animal Light". The mistake is not, then, a misprint, for otherwise one cannot conceive how a physiological work (dealing particularly with the lower organisms) could be listed where it is. Again, on page 335, amidst the vertebrate works we suddenly come on a volume by Exner

[1] "An Introduction to the Literature of Vertebrate Zoology", compiled and edited by C. A. Wood, M.D., LL.D.; 1931. London: Humphrey Milford (Oxford University Press). Demy 4to., pp. 662. Price: 3 guineas.

on the compound eyes of insects and crustaceans, a very curious stray indeed. Dozens of similar examples could be quoted. The hotch-potch is at times extraordinary. Figures are also carelessly stated. The Challenger Expedition is dated 1881 on page 32, whereas it should be 1872-1876. The Maldive and Laccadive Islands are considered to be part of Australasia, and so on.

It is most difficult to understand how the opportunities afforded in this volume have been missed. It is more surprising that we can say the volume has its uses, but its purchase will be more by libraries than by private individuals.

SEX LIFE.

A BOOK of three hundred pages, entitled "The Sexual Life of Man", is described on the paper wrapper as an outline for students, doctors and lawyers.[1]

The fact that this manual has reached a second edition indicates that it fulfils a need by supplying information for which there is a demand. In any book on the sexual life of man adequate space should be given to consideration of normal sexual function; this, however, has not been done. The book also reflects an unscientific bias on the psychological side which reduces its value very considerably.

The author has rightly paid a great deal of attention to endocrine factors, with special reference to the theories and experiments of Steinach. Twenty-seven pages are devoted to an aspect of sex that is seldom ventilated in books on the subject—forensic problems.

Masturbation is discussed in a sane manner, the author's attitude recalling that of Havelock Ellis and other authorities. The author quotes Kretschmer's description of masturbation: "a harmless and transient physiological by-product of the healthy sexual impulse, seemingly a safety-valve, more especially when sexual intercourse encounters one of the many barriers necessary in civilized communities". It is true that he regards "excessive onanism" as having "a temporary weakening effect on the spinal sexual centres", but he even mentions many conditions in which he regards moderate masturbation as actually beneficial. Such opinions he recognizes to be directly opposed to what he calls "distorted, foolish ideas of the supposed injurious after-effects of masturbation culled from some wretched book".

On page 164 he states: "It (masturbation) affects 99 per cent. of all young men and girls, and one might safely say that as far as men are concerned every adult man was once an onanist—even mature men and women are quite frequently masturbators, even during their whole life-time". This paragraph constitutes an over-statement, no doubt, but it is the author's intention to state forcibly that moderate masturbation does not produce physical harm. Also he wishes to emphasize the belief that when after-effects do occur, they are the result of "foolishly alarmist literature".

Unfortunately, the author fails to realize what is the reason that alarmist views are held by many moralists, and why neurotic subjects are so little reassured by rational opinion. Understanding of these points (as of many other problems of sex) can only follow realization of the maladjustments to reality that are revealed by psycho-analysis, a method of investigation and treatment upon which the author of this book expends so much invective.

HISTOLOGICAL METHODS.

J. T. HOLDER'S little book, "Elementary Histological Technique for Animal and Plant Tissues", is stated by the author to be intended for the beginner working independently, not for the skilled investigator.[1] As such it fulfils its purpose very well, but still leaves room for a volume of equal size to which the term admirable can be applied.

The writer first describes the essential apparatus required for staining and section cutting (paraffin and celloidin methods) and the media for mounting histological preparations. Following on this is a chapter on the examination of fresh tissues, instructions being given for the dissociation *et cetera* of nerve cells and epithelial cells, and for the preparation and staining of blood smears. Then the process of section making is taken step by step from the first stages of fixation to the final staining and mounting. Only the most important and generally used fixatives are described. This part is well stated, clearly and without waste of words. It is simple, but practical. Our only criticism is that in describing the difficulties met with by the beginner the author might quite well have referred to "electrification", a frequent nuisance which puzzles the tiro.

The last chapters of the book are concerned with the same matters as applied to botanical material. The combination of the two sides is useful, for many methods invented by anatomists, zoologists and botanists are of general application.

The book is most suited for the university student taking senior courses which involve microscopical technique in plant and animal histology. It must not be regarded as a work of reference for any of the more specialized technical methods.

ANOTHER BOOK ON GENERAL PRACTICE.

"A MANUAL OF GENERAL PRACTICE", by W. Stanley Sykes, which has somehow reached a second edition, is one of those books intended for the guidance of the newly qualified.[2] The contents consist chiefly of laboured platitudes, puerile statistics and quite needless lists of drugs, equipment and certificates.

The type of practice engaged in by the author is foreign to Australia, but the description of it may interest any who contemplate a visit to England and who, for one reason or another, may possibly practise in the industrial parts. We hope that they will tell us of their experience of pneumonia there. Respiration rates of 90 to 100 per minute are apparently not out of the way and are not necessarily of evil omen. They must remember to collect the twopence for the medicine bottle, since 600 bottles last only a few weeks in a Yorkshire practice.

Notes on Books, Current Journals and New Appliances.

ANOTHER BOOK ON RUSSIA.

MRS. E. G. GRADY, whose husband was employed by the Soviet Government in Russia, has written an account of her experiences and of her impressions.[3] She gives an interesting account of the way in which foreigners employed in Russia are compelled to live, and the picture painted by her of the sufferings of the Russian people is most vivid. She describes the camouflage designed to hide the truth from the outside world. At the same time she has been able to see the funny side of much of the Soviet propaganda and ends her book with a laugh. The laugh, in our opinion, is forced, for there is too much misery and suffering mixed with the ludicrous.

[1] "The Sexual Life of Man, An Outline for Students, Doctors and Lawyers", by Dr. Placzek; Second Edition; translated by L. S. Morgan, M.R.C.S., L.R.C.P.; 1931. London: John Bale, Sons and Danielsson.

[1] "Elementary Histological Technique for Animal and Plant Tissues", by J. T. Holder, F.R.M.S.; 1931. London: J. and A. Churchill. Demy 8vo., pp. 111, with 23 illustrations. Price: 7s. 6d. net.

[2] "A Manual of General Medical Practice", by W. S. Sykes, M.A., M.B., B.Ch., D.P.H., M.R.C.S., L.R.C.P.; Second Edition; 1931. London: H. K. Lewis. Crown 8vo., pp. 225. Price: 7s. 6d. net.

[3] "Seeing Red: Behind the Scenes in Russia To-day", by E. G. Brady; 1931. New York: Brewer, Warren and Putnam; Australia: Angus and Robertson. Crown 8vo., pp. 257. Price: 6s. 6d. net.

The Medical Journal of Australia

SATURDAY, JANUARY 30, 1932.

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OBSTETRICS.

PROBABLY more has been written about obstetrics than about any other subject in the medical curriculum. The teaching of students has been discussed almost *ad nauseam*, and methods of practice have been subjected to searching inquiry. Discussion and inquiry have been all to the good if they have made medical practitioners think about the subject. Thinking, however, is not sufficient. While numbers of practitioners have made every effort to improve their practice of obstetrics, there are still many who give only perfunctory antenatal supervision, who object to being called to the bedside before the head of the fœtus is on the perineum, and who deliver the child by forceps in almost every instance. There is still need to press for improvement in the standard of obstetrics.

The standard of obstetrics may be improved in several ways. The first is by securing adequate undergraduate and post-graduate teaching. In all Australian universities and in the University of New Zealand much thought has been given to this matter, and chairs of obstetrics have been established in each university with the exception of the University of Adelaide. Throughout the whole of Australasia the teaching of obstetrics to students is well organized and thorough, and no student at graduation can plead ignorance as a cause for deviation from the path of obstetric righteousness. Although the arrangements for post-graduate teaching are not so extensive as those for undergraduates, it is undoubtedly true that no medical practitioner who wishes to study obstetrics once again from either its theoretical or its practical aspect, is unable to achieve his objective. A second way in which the standard of obstetric practice may be raised is by administrative or departmental activities. In several States the Health Department has its division devoted to maternal and child welfare and makes inquiry into deaths from puerperal fever. Whether these provisions are effective or not need not be discussed at present. Suffice it to state that legislation, however necessary it may be, does not always, more is the pity, produce cooperation between departmental officer and private practitioner. This was shown in an article on obstetrics in New Zealand by Professor Marshall Allan, published in the issue of January 9, 1932. A third method, which may be described as consultative or investigational, is likely to produce the best results if it has been preceded by adequate teaching. Those who adopt this method keep accurate and complete records of their cases and meet at regular intervals to report their results, discuss their methods and investigate the reasons for their failures. For the past few years the Obstetrical Section of the Queensland Branch of the British Medical Association has followed this plan. The primary object of the Section was to collect records of two thousand consecutive midwifery cases occurring in the private practices of members. This objective has been gained and in another place in this issue there appears the report of the Section on these cases. Readers are asked to read this report carefully and to note in the prelude to the report the details which are recorded in every instance.

There can be no doubt that this method of working is most desirable. It is likely to be productive of good chiefly because it is undertaken voluntarily. Further, by a discussion of failures errors in technique will be discovered and mistaken judgements will be brought to light. Most medical practitioners

prefer to advertise their successes and would gain glory for themselves by a stroke of fortune which brings a patient with a rare pathological condition within their sphere of action, when they perhaps do no more than assist the *vis medicatrix naturæ*. Moreover, a frank discussion of failures will go far towards eliminating professional jealousy, that green-eyed monster, which, like the jealousy of lovers, scatters common sense to the winds and makes men and women look ridiculous. Professor Marshall Allan, in his address to the Victorian Branch Section, advocated the adoption of this method of investigation and referred to its success in New Zealand. We commend the Queensland report to all the Branches and would urge the Queensland Section not to be content with its report of two thousand cases. Its members have put their hands to the plough; to be content with one harvest would make them unworthy.

Current Comment.

BETEL-NUT CANCER.

MATTHEW J. STEWART, in the Croonian Lectures,[1] dealt exhaustively with precancerous lesions of the alimentary tract. Among other lesions he discussed betel-nut cancer of the cheek. The incidence of carcinoma of the cheek is remarkably high in some districts of India and the East, where the habit of betel chewing is widespread. In southern India A. Fells noted that of 377 cases of epithelial cancer seen by him, 91·5% were connected with the buccal cavity, 83·2% of these originating in the cheek, 9·3% in the tongue, and 7·5% in the lip. The "quid" of betel is usually carried inside the cheek, and changes occur in the buccal mucosa at this point, just opposite the molars and premolars; the changes eventually become malignant. Inflammatory thickening and roughening of the mucosa, leucoplakia and epithelial denudation supervene, followed by progressive ulceration. The incidence of cancer varies greatly in different betel chewing populations. Betel chewing is common in the Dutch East Indies, but no evidence has been adduced that betel cancer is frequent there. The same applies to Formosa and Guam. Degorce saw only few cases in Tonkin, whereas Montel noted many oral cancers in Saigon, in a similar Indo-Chinese population. The following Indian figures have been compiled: In Travancore, of cancers in all sites, 38% were buccal; in Madras 42·1% and in Ceylon 32% of all cancers were buccal. The exact composition of the "quid" varies in different countries and in different districts, and it has been suggested that the varying cancer incidence may be due to this difference. It has been suggested that in Madras tobacco chewing in addition to betel chewing is responsible for the high incidence, and the short, strong cigar used by the natives of Ceylon has been blamed for the incidence in the Cingalese. Betel-nut cancer affords an excellent example of the importance of local factors in cancer causation, ranking with "Kangri" cancer, tar and soot cancer and mule-spinners' and X ray cancer.

Much has been written in recent years concerning betel chewing and betel cancer, and the matter is of considerable interest to Australia from the fact that betel chewing prevails in many parts of Papua, the Mandated Territory of New Guinea, including New Britain, the Solomon Islands and many other islands in the North and South Pacific. In all these areas, however, betel cancer is either rare or non-existant. The usual betel quid consists of a pepper vine, "pan" or "sireh" (*Piper betle*), the scrapings of the seeds (or nuts) of a palm (*Areca catechu*) and slaked shell lime, which is said to effect the slow liberation of the active ingredients during mastication. According to A. Dickson Wright, in 1924 45% of deaths from cancer in Ceylon were due to growths of the cheeks, jaw and lips. Squamous-celled carcinoma appears in the sulcus between the lower jaw and the cheek, just where the betel quid is stored. In Ceylon the quid includes also the pharmacopœial *Catechu pallidum* (*Uncaria gambier*). The cancers are very malignant and rapidly perforate the cheek, becoming beyond operative control. The carcinogenetic number of the different ingredients has not been decided. According to A. G. Ellis, in Siam the ingredients are betel pepper, areca nut, black catechu (*Acacia catechu*), lime, turmeric and tobacco leaf. In Siam betel nut chewing is more nearly universal among women than men, and generally women chew more of it. In the addict the tongue, lips and buccal mucosa become brownish red and the lips and tongue roughened. The teeth become dark brown or nearly black. Lime is deposited on them, preventing decay. The gums recede, pyorrhœa is common, the alveolar processes atrophy, and the teeth loosen. Betel nut chewing does not seem to have any antiseptic effect. In a Bangkok hospital, of operations performed for malignant disease during six years, half of the number were of the lip, 56% being in men and 44% in women, as compared with the usual 90% incidence in males generally. R. L. Spittel has admitted that the number of betel cancers is very small in comparison with the thousands who indulge in the habit of chewing, but such cancers are sufficiently numerous to indicate something more than coincidence. Cancers of the cheek are among the commonest malignant growths encountered in Ceylon, being equally distributed between the sexes, just as betel chewing is indulged in by both sexes. Spittel inclined to the opinion that the lime provided the continuous irritant, but that the potency of the crude tobacco could not be overlooked.

[1] *The Lancet*, September 12, 1931.

Cancer of the cheek, as seen in Ceylon, nearly always begins as a leucoplakia or roughening of the buccal mucosa in contact with the molar and premolar teeth, which are discoloured and coated with rough deposits of tartar and lime, displaying a surface like sandpaper. Archibald Leitch made the pertinent suggestion that epithelioma of the inside of the cheek might be no more common in betel chewers than in populations not so addicted; at the same time he did not deny the possibility of betel chewing being the cause. Snijders, in 1923, asked why cancer of the buccal cavity should be so common in Travancore and Saigon and much rarer in Tonkin and the Dutch Indies, with betel chewing common in all those countries. J. L. Maxwell observed that in South Formosa, where he practised for nearly twenty years, betel chewing was common, the mixture consisting of betel nut, a small piece of catechu, lime and the vine leaf. Buccal cancer was a rarity. He did not know whether to ascribe such rarity to racial immunity or whether there was some unknown factor in relation to the habit, operative in India, but absent from Formosa. Sarcoma of the duodenum has been reported in a Siamese betel chewing woman.

There can be no doubt that the habit of betel chewing is followed in many cases by buccal malignant disease, but only in certain topographical areas. Whatever ingredient or combination of ingredients determines the malignant change, geographical and possibly racial factors are of very great importance. In considering the individual constituents in order to ascertain which might produce continuous or repeated irritation, lime occurs first to the mind. Quicklime (calcium oxide) is caustic and irritant, but slaked lime (calcium hydroxide) is also, though to a lesser degree, irritant. Calcium hydroxide, however, would be more or less rapidly converted into calcium carbonate or chalk, which is sedative and astringent. The betel (areca) nut has a slightly acrid and astringent taste. In addition to its employment as a masticatory it has been used in dentifrices. It contains a fixed oil, tannic acid and three alkaloids, one being volatile. It is sialogogue and astringent and is in common use as an anthelmintic in man and dogs, particularly for tapeworm. The areca palm is indigenous in Northern Queensland, but there is no record of the aborigines employing it as a masticatory. Betel pepper is also much in use as a masticatory and sialogogue. In action it closely resembles the ordinary black pepper of the household. The leaves have a burning, bitter, aromatic taste, due to a volatile oil which contains betel-phenol, isomeric with the antiseptic eugenol. In Brumer Island, south-east of Papua, where both sexes indulge in the habit, the green fruit of the betel pepper vine is used as well as the leaves. The volatile oil might conceivably provide a repeated irritant to the buccal mucosa. Similarly, when turmeric (*Curcuma longa vel rotunda*) is included in the mass, its volatile oil might exert an irritant action. Turmeric, however, is used as a condiment in India and China. *Catechu pallidum* and *Catechu nigrum* are both essentially tannic acid preparations repeatedly used as local and intestinal astringents. *Catechu pallidum* contains also Gambier-fluorescein, but *Catechu nigrum* has no fluorescent body. As regards tobacco, no record can be found of local malignant disease in the snuff-takers or tobacco chewers of old. An exhaustive examination of betel chewers by the medical officers of Papua and the Mandated Territory of New Guinea might yield interesting information regarding the incidence of malignant degeneration, if any, together with the exact composition of the betel mass.

THE LATEST SPECIALTY.

AMERICA has been described as the home of specialization, and it is certainly true that specialists in American institutions are easy to find. Australians working with Americans in France during the war used to look for American medical officers who were not specialists. While it must be ceded that medicine owes much to America because of the concentrated efforts of her investigators and practitioners along one channel, specialization can go so far as to become absurd. The latest absurdity comes from T. H. Morrison and S. Morrison, of the Department of Medicine, University of Maryland.[1] These investigators are attached to a "gastro-enterological clinic". To say that a man is a gastro-enterologist and that he devotes his time to the study of the alimentary tract and its diseases is, in our opinion, going too far with specialization, but T. H. and S. Morrison are not content. They see a neurotic element in gastric disease; they are not satisfied that a gastro-enterologist can treat the patient satisfactorily—he would look at the gastric condition and neglect the neurosis. On the other hand, they doubt the ability of a psychiatrist to pay due attention to the gastric condition. They have therefore introduced a new specialty—gastro-psychiatry! They write:

> We have introduced the term gastro-psychiatrist. The temperamental fitness of this individual differs greatly from the average psychiatrist or gastro-enterologist. He stresses neither the "whole" of the former nor the "part" of the latter. He takes both into consideration *equally* and does not hesitate to cure the part while treating the whole. Not every gastro-enterologist and psychiatrist is suited for this specialty; they may practice (*sic*) it, but not with all attainable success.

Part of this quotation is, of course, quite unintelligible, and the views of T. H. and S. Morrison are unimportant. What does matter is that medical practitioners do not always pay attention to the mental element in disease. The mental aspect of gastric disease has been emphasized in this journal by S. F. McDonald and by John Bostock. Not only is it necessary to recognize the existence of a mental factor, but this factor must be treated; if it is ignored, the patient will probably not be cured of his complaint. If the mental factor in gastric disease is treated, there will be no need for such over-specialized persons as "gastro-psychiatrists".

[1] *Annals of Clinical Medicine*, September, 1931.

Abstracts from Current Medical Literature.

PÆDIATRICS.

The Allergic Child.

BRET RATNER (*The Journal of the American Medical Association*, February, 1931), in discussing the allergic child, stresses the importance of a carefully compiled family and personal history, extending back to infancy. In this history inquiry must be made into the occurrence of infantile eczema, including the time of day and exact period at which it first appeared. From animal experiments the author concludes that a high protein diet during the mother's pregnancy may have led to sensitization; this point is investigated and confirmation sought by means of skin tests. The home and its environments should be examined in order to discover the source of any antigenic dust to which the patient may be subjected, and the wage-earner's occupation analysed for a possible antigenic vector. An important factor in making skin tests is the activity of the proteins used. A patient, for example, may react to an extract from feathers from his own pillow, but not to a stock extract. The author favours the scratch method in children as being simpler, less painful and safer than the intradermal injection. Great care should be taken not to produce trauma, as blood prevents the protein from entering the lymphatics. A general concept must be formed of the skin sensitivity as a whole in order to decide whether a given wheal may be read as indicating a reaction. Not all skin areas are equally sensitive, and after a doubtful response many different areas can be tried. In intestinal allergy a skin reaction is not always obtained. A complete diagnostic procedure includes testing with extracts of all the available proteins, with extracts of all dust producing substances in the home and with occupational substances in and out of the home; full chemical and cytological investigation of the blood must be made, and radiograms of the chest and sinuses must be taken. Dick, Schick and Mantoux tests must be made and complete urinary examination carried out. In management, elimination should be personally supervised, and all eventualities considered. Certain foods or pillows should be recommended, as otherwise parents will substitute an equally potent antigen. Denaturization of foodstuffs by prolonged heating may render the essential foods available and prevent serious nutritional sequelæ, or specific desensitization may be undertaken by drop doses by mouth or very cautious injections of great dilution. For simple food sensitization in infancy the best method is undoubtedly elimination *plus* oral desensitization, but severe multiple desensitization requires years of patient care and work, in which elimination must play the larger share, and later, careful individual desensitization, but this is better than the frequently failing short-cut by way of non-specific measures.

Extrasystoles in Childhood.

M. LEFFKOWITZ (*Klinische Wochenschrift*, August 22, 1931) has investigated the effect of extrasystoles in a group of twenty-six children. In only two cases had the children complained of symptoms which caused cardiac investigation. As a result of this survey he is of the opinion that extrasystoles may be noted equally among children with sound as well as diseased hearts. As with older persons, subjective symptoms are not common. Ventricular extrasystoles are more frequent than those of auricular origin. The former group have no effect on the development of the heart. In a small percentage of the cases of auricular extrasystole, however, serious damage to the heart was observed. On the whole, treatment during childhood is not advised. The majority could perform gymnastic exercises as well as healthy children, and the reaction to exercise seemed to be very good.

Treatment of Anæmia in Infants.

E. SCHIFF AND N. JOFFE (*Klinische Wochenschrift*, October 17, 1931) discuss the treatment of anæmia in premature infants, with particular reference to the value of copper. At first iron and copper were used in alimentary anæmia, but the results were not so good as when copper alone was given. They used, twice a day, ten to twenty drops of a 1% solution of copper sulphate. In general, however, copper cannot absolutely prevent this and other indefinite varieties of anæmia, and further investigations are needed to establish the exact causation of these anæmias of the premature infant.

Spinal Tumours in Childhood.

J. CRAIG AND A. MITCHELL (*Archives of Disease in Childhood*, February, 1931) record two cases of spinal tumour, a cholesteatoma and a tuberculoma, for both of which successful operation was performed. The tuberculoma occurred in a girl, aged eight years; root pains had been present for eighteen months, after which the power of walking gradually decreased. She presented a lumbar lordosis, with pain in this region when flexing the head forward and on deep percussion over the last dorsal spines. She was analgesic below the umbilicus, where a band of hyperæsthesia was present. Plantar and organic reflexes were normal, but the knee jerks were absent and the deep sensibility of both legs impaired. A radiogram of the spine revealed no abnormality, and no evidence could be found of a tuberculous infection. Froin's syndrome was present and the Queckenstedt test gave a positive reaction. Operation in two stages disclosed a thickened vascular cord with nothing sufficiently localized for removal. A fragment of the vascular tissue proved it to be a spinal tuberculoma. The girl recovered completely as a result of the simple decompression followed by four exposures to the deep X rays. The other patient, a boy of eight years, had had pain in the lumbar region for three years, also associated with lordosis and made worse by flexing the head. For a year he had had nocturnal enuresis, was tender to deep percussion over the twelfth dorsal vertebra. A radiogram revealed no abnormality and a lumbar puncture showed a great increase of globulin in the cerebro-spinal fluid. Operation in two stages revealed a motionless dura below the eleventh dorsal vertebra and later a pearly white tumour which proved to be cholesteatoma. Recovery was uneventful, the only residuum being a slight weakness of extension at the left knee. Spinal tumours are rarer in childhood. Sixteen occurred under the age of fifteen in a composite series of 381 patients submitted to operation. Tuberculomata are probably not so common as was once supposed.

Blood Phosphorus and Calcium.

MILAN SOKOLOVITCH (*Archives of Disease in Childhood*, June, 1931) has investigated the phosphorus compounds and calcium in the blood of children. He included blood of normal, nephritic, diabetic children and a miscellaneous group. He finds that in disease the total phosphorus in the blood is not increased, but merely redistributed. The direction of change in any fraction may be either an increase or decrease, but of the three fractions, free, ester and lipin, the ester phosphorus showed the greatest stability. There was a general tendency for the total phosphorus to fall in amount during disease.

Post-Anginal Sepsis.

MARK S. REUBEN (*Archives of Disease in Childhood*, April, 1931) gives the title to "sepsis of oro-nasopharyngeal origin" in a lengthy discussion of its various features, including a possible termination by thrombosis of the internal jugular veins. The condition is apparently better recognized in Germany than in America or England. The process is apparently a septic cellulitis of the neck following, after a latent period, sore throat, tonsillitis, tooth infection, sinus disease, otitis, adenoiditis *et cetera*. Hypertrophy of lymph glands is the first and mildest manifestation, but may pass into phlegmon and thence by gravity to the mediastinum or jugular veins, whence septic emboli can enter the lungs, or retrograde thrombophlebitis involve the intracranial venous sinuses or meninges. Pressure may occur on the trachea, œsophagus, carotids or vagus nerve, or the carotids may be actually eroded or thrombosed. Ten cases have occurred in children under ten years in the Beth Israel Hospital, New York, in less than eighteen months. Diagnosis rests upon the history of a primary infection, the latent period,

followed by constitutional signs of sepsis, usually an external swelling on the neck or signs of metastasis. Acute leuchæmia, glandular fever, Vincent's angina and diphtheria require exclusion. Surgical treatment consists in early incision of the external swelling. This, with active treatment of the primary focus, may obviate the necessity for tying the internal jugulars later, should they become palpable or should signs of extension to the thorax or cranium appear.

ORTHOPÆDIC SURGERY.

Extracapsular Fractures of the Femur in the Aged.

AUGUST WENDEL (*The Journal of Bone and Joint Surgery*, July, 1931) contends that the Russell treatment is the simplest and most efficient described method for fractures of the femur in its upper two-thirds outside of the joint capsule. He considers that if the surgeons who are unfamiliar with the method, were aware of the accurate traction that the Russell apparatus exerts in the longitudinal axis of the femur, and were to give it due trial, its popularity would greatly increase, and direct skeletal force would not be so popular in the treatment of similar fractures in this region. He also considers that the introduction of the Wilkie apparatus into the Russell treatment better adapts the latter to extracapsular fractures in the aged, as pulmonary complications are reduced to a minimum. This reduction of complications is due to two reasons, namely, it is no longer necessary to lie flat on the back for six weeks, and secondly, after three weeks and until six weeks after the injury, while in the Wilkie apparatus, the patient may be shifted in bed to any position in the horizontal plane. After the seventh week consolidation is usually completed in aged patients and the Wilkie apparatus is removed.

Periarterial Sympathectomy in Fractures of the Lower Extremity.

R. COLP AND S. MAGE (*The Journal of the American Medical Association*, October 10, 1931) report results of periarterial sympathectomy in a series of seven recent fractures of the lower extremity and in ten cases of delayed union, the latter of which were ununited after an average of seventy days. In the recent fractures clinical union was obtained in eleven days less, and in a similar series the patients remained on an average nineteen days less in hospital. In the cases of delayed union, clinical union resulted twenty-one days after sympathectomy in eight instances.

Thrombo-Anglitis Obliterans.

N. W. BARKER (*The Journal of the American Medical Association*, September 19, 1931) describes results obtained from treatment by foreign protein of *thrombo-angiitis obliterans* in various groups, namely: Group I, characterized by claudication without pain while the patient is at rest, or trophic changes; Group II, characterized by pain while the patient is at rest, without ulcers or gangrene; Group III, characterized by simple ulcers and pain during rest; Group IV, characterized by pain during rest, and gangrene limited to the digits; Group V, characterized by massive gangrene extending proximally to the digits. The results in all groups show marked improvement in 49%, moderate or slow improvement in 27%, no improvement in 6%, and progress of the disease in 18%. The foreign protein used was typhoid, paratyphoid *A* and *B* vaccine ("T.A.B.") and typhoid "H" antigen". It was claimed for the latter that it tends to cause less chill and malaise for the amount of fever produced as compared with the former, but that it has about the same therapeutic effect. The author claims that this method of treatment tends to relieve pain and to give increased blood supply to the extremities, thus accelerating the healing of open lesions. It has relatively very little effect in patients in whom there is claudication only or when extensive gangrene is present.

Angiospastic Syndromes in Extremities.

J. J. MORTON AND W. J. M. SCOTT (*Annals of Surgery*, November, 1931) conclude that there are several types of angiospasm which can be recognized and separated into major groups as follows: (*a*) That occurring in organic vascular disease, (*b*) idiopathic paroxysmal (Raynaud's disease), (*c*) that dependent upon organic or functional nervous diseases, (*d*) that consecutive to trauma, (*e*) venospasm. In Raynaud's disease the fundamental abnormality is a hypersensitivity of the peripheral arteries to cold. However, vasoconstrictor impulses play an important rôle by initiating and accentuating many of the attacks. Consequently, the advisability of removing the sympathetic innervation can be determined by the effect of regional anæsthesia in releasing the spasm during an attack. They also contend that organic and functional nervous disorders are frequently accompanied by an accentuated local vasoconstrictor tone. Trauma in the extremities may be followed by an arterial spasm, frequently associated with pain. This is probably due to vasoconstrictor impulses induced by reflex afferent stimuli from the traumatized area. They believe that such a reaction is a fundamental response incident to trauma and scar formation. There is an individual variation in the degree of manifestation of this reaction, and it may be present as a latent hypersensitivity to cold. Evidence is presented that angiospasm, affecting principally veins, occurs as a clinical entity.

Transplantation of Bone.

IN a discussion on transplantation of bone in *The Lancet*, September 26, 1931, W. E. Gallie described the indications for and precautions necessary in performing transplantation of bone. He emphasized his belief that the transplant dies and ultimately undergoes complete absorption in its new growth. Success in transplantation depends on, first, primary union, secondly, firm healing of graft to bed, and, thirdly, final union of fragments to one another. The commonest cause of failure is not securing contact of graft to bed over a sufficient area. For example, in radius or ulna a graft should be embedded 3·75 centimetres (one and a half inches) into each fragment, and in the tibia 6·25 centimetres (two and a half inches) at each end. The contention that close apposition between graft and bed is all important is supported by experimental work. Subsequent immobilization is of supreme importance and necessitates internal fixation as well as an external splint of plaster of Paris. It is important to remove the sclerosed ends of the bone concerned, although Gallie does not agree with the contention of some writers that the mere removal of sclerosed ends and free opening up of healthy bone are sufficient to secure union. He disagrees with the medullary peg, as it too often acts as a plug, preventing the necessary outflow of osteogenetic tissue from the bone ends. The graft itself must contain living elements. It must be strong enough to act as a temporary splint and must resist disintegration until sufficient new bone had been laid down to prevent its collapse. It should contain both endosteal and subperiosteal cells, but the periosteum need not be taken with it, since, when left behind, it can be sutured to close the cavity from which the graft came.

Treatment of Chronic Arthritis by Blood Transfusion.

W. S. C. COPEMAN (*Proceedings of the Royal Society of Medicine*, September, 1931) describes sixteen instances of arthritis treated by transfusions of five hundred cubic centimetres of whole blood. He claims that these transfusions were benficial in cases of rheumatoid arthritis and infective arthritis, but in chronic osteoarthritis there was no definite benefit. One of the directions in which improvement resulted was that vigorous treatment was possible a few days after injection. Joint pains improved, and the improvement included benefit to the general health and outlook of the patient.

Still's Disease and Rheumatoid Arthritis.

RUPERT WATERHOUSE (*Proceedings of the Royal Society of Medicine*, October, 1931) describes several cases of rheumatoid arthritis occurring in adults, which were clinically indistinguishable from cases recognizable as Still's disease in children. He claims that there is no essential difference, either clinically or pathologically, between the two conditions, and that the ætiology of both is equally obscure.

British Medical Association News.

SCIENTIFIC.

THE OBSTETRICAL SECTION OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION has issued its report on two thousand consecutive midwifery cases occurring in the private practices of its members. This report is referred to in the leading article in this issue. The report is as follows.

REPORT ON TWO THOUSAND CONSECUTIVE MIDWIFERY CASES.

Preamble.

In 1928 it was decided to form an Obstetric Section in connexion with the Queensland Branch of the British Medical Association. The primary object of the section was to collect records of two thousand consecutive midwifery cases attended by members in their ordinary private practice in the city of Brisbane. (Cases attended at the public hospital were not eligible for inclusion.)

The second object was, by holding regular quarterly meetings, to promote the study of obstetric procedure, by discussing the management and treatment of all the cases of interest reported during the previous quarter.

In order to attain the primary object, the members agreed to fill in the forms drawn up by the Executive of the Section, and the Statistical Subcommittee was thus supplied with the following information about each case.

Age. Parity. Nationality of each patient.
Any previous obstetric history.
Date of last menstrual period.
External pelvic measurements.
Antenatal observations and treatment. Urine. Blood pressure *et cetera*.
General condition of the patient.
The course of the confinement.
Whether induction was resorted to; if so, whether it was medical or surgical.
Time and date of the commencement of labour.
Presentation on delivery.
Mode of delivery: natural, instrumental or otherwise.
Length of the three stages of labour.
Condition of the child at birth: alive, macerated or stillborn. (In the last 1,000 cases a note has been made of any child dying during the puerperium.)
Weight. Approximate age. Any abnormality.
Any laceration of the perineum (first, second and third degrees, episiotomy.)
Mode of expulsion of the placenta: natural, pressure, manual removal.
Maternal mortality.
Maternal morbidity. (Temperature of 100·2° F. or more for twenty-four hours or on more than one occasion.)
Lactation: natural, supplemented or artificial.
Location: Whether patient was attended in her own home, in a private maternity hospital or in a private general hospital, taking any medical or surgical as well as maternity cases. (In the last 1,000 cases a record has been taken of cases attended in their own homes as to whether a whole or part time nurse had been engaged.)

Space was left for a post-natal examination six weeks or more after the confinement, but this has been so seldom filled up that it is now omitted.

In any case of abnormality the member was requested to amplify these headings, with full details, treatment, temperature charts *et cetera*.

From the records supplied the Statistical Subcommittee has made the following analysis.

Analysis of Two Thousand Cases.

The two thousand cases are analysed in Table I. Table II shows the lacerations of the perineum, Table III shows the environment.

TABLE I.

Characteristic Feature.	First Thousand.	Second Thousand.	Total Two Thousand.
Maternals deaths	5	3	8
Still-births	25	20	45 } 66
Macerated fœtuses	13	8	21 }
Plural births	12	13	25
Eclampsia	7	5	12
Albuminuria	83	53	136
Pyelitis	22	12	34
Placenta prævia	5	5	10
Ante partum hæmorrhage	9	8	17
Post partum hæmorrhage	21	17	38
Puerperal morbidity	85	61	146
Occipito-posterior presentations	90	86	176
Including cases delivered as persistent occipito-posterior presentations	26	29	55
Breech	32	26	58
Face	1	1	2
Brow	0	1	1
Hydramnios	3	4	7
Prolapse of cord	5	4	9
Succenturiate placenta	2	0	2

TABLE II, SHOWING LACERATIONS OF THE PERINEUM.

Degree.	First Thousand.	Second Thousand.	Total.
First degree	162	163	325
Second degree	80	83	163
Third degree	1	4	5
Episiotomies	15	11	26
Total	258	261	519

TABLE IV.

Condition.	First Thousand.	Second Thousand.	Total.
Severe sepsis	3	4	7
Mild sepsis	33	19	52
Sapræmia	10	9	19
Mastitis	19	17	36
Phlegmasia alba dolens	1	1	2
Acute necrosis of liver	1	0	1
Eclampsia	0	1	1
Pyelitis	6	1	7
Influenza	2	3	5
Bronchitis	1	1	2
Constitutional	1	0	1
Unclassified	6	5	11
Suppurating fibroid	1	0	1
Bartholins abscess	1	0	1

TABLE III, SHOWING ENVIRONMENT.

Location.	First Thousand.			Second Thousand.			Total.		
	Number	Morbidity	Per-centage	Number	Morbidity	Per-centage	Number	Morbidity	Per-centage
Home	363	22	6·0	357	12	3·08	720	34	4·54
Maternity home	478	45	9·4	404	23	5·69	882	68	7·54
General hospital	152	17	11·1	234	7	2·99	386	24	6·54
Not stated	7	0	0	5	0	0	12	0	0

Instrumental deliveries numbered 230 in the first thousand, 239 in the second thousand, a total of 469.

Puerperal morbidity is classified as in Table IV.

Notes on Eight Maternal Deaths.

1. Natural delivery occurred after a labour of forty-one hours. A first degree laceration of the perineum occurred. Pelvic cellulitis supervened. Operation was performed and the patient died on the tenth day.

2. Natural delivery occurred. An adherent placenta was manually removed. Death occurred from septicæmia on the thirteenth day.

3. The patient had previously had one child. Eclampsia occurred the fourth day after delivery. The patient became comatose and died on the twelfth day. Antenatal treatment for albuminuria had been carried out.

4. The patient had previously had one child. She was thirty-two years of age. The cord prolapsed; the presentation was persistent occipito-posterior, and delivery was instrumental. A second degree laceration resulted. Labour lasted fourteen hours. The placenta and membranes were complete. The patient died of septicæmia twenty-one days after the birth of the child. She had received antenatal treatment for albuminuria.

5. Acute necrosis of the liver; death occurred from coma seventy-two hours after delivery. Diagnosis was confirmed by histological examination of the liver.

6. Death followed concealed accidental hæmorrhage.

7. Death occurred from septicæmia following manual removal of the placenta.

8. The patient was a very debilitated woman, and death occurred from *post partum* hæmorrhage.

Cæsarean Section.

Cæsarean section was performed four times.

1. The operation was performed for *placenta prævia* in a *primipara* aged thirty-five years. She suffered from repeated hæmorrhages *per vaginam* during the seventh month. As the patient's condition was becoming serious, it was decided to perform Cæsarean section during what was estimated to be the thirty-third week. The result was good as far as the mother was concerned. The child died.

2. The operation was performed for eclampsia. The result was satisfactory for both mother and child.

3. The operation was performed for disproportion after a trial labour. The result was successful for both mother and child.

4. The operation was performed for disproportion after a trial labour. The result was successful for both mother and child.

External Version.

External version has been reported fourteen times, to convert a breech into a cephalic presentation.

Still-Births.

The 66 still-births reported include 21 cases in which the fœtus was macerated, so that the correct figure for still-births is really 45. Of these seven were preceded by *ante partum* hæmorrhage, two were preceded by eclampsia; three were preceded by severe maternal albuminuria; in four various deformities of the fœtus were present; five were complicated with prolapse of the cord; two followed internal version; six were breech deliveries; eight deliveries were instrumental, with prolonged labour; in one a contraction ring was present; and in seven the cause was not classified.

The two internal versions were performed in cases which were expected to be difficult confinements.

Of the six breech deliveries, four were in *primiparæ*. Of the two still-births in *multiparæ*, in one the arms and legs of the fœtus were extended, and the other was complicated by a prolapsed cord.

Short notes of the instrumental deliveries with still-born fœtuses are appended.

Number 19.—Occipito-posterior presentation in a *primipara*. Premature rupture of the membranes occurred. Rotation was effected manually and the child was delivered by instruments.

Number 83.—A *primipara* had a labour lasting four days. The presentation was left occipito-anterior. Delivery was instrumental. The placenta was fibrotic.

Number 230.—A large child, weighing eleven pounds, was delivered by instruments. There was difficulty with the shoulders. This was a case of disproportion. The patient's first child was still-born, and with the second labour had been induced at three and a half months.

Number 490.—Labour was long, lasting forty hours. The position was occipito-posterior. Delivery was instrumental. The cord was round the child's neck.

Number 604.—Labour was long, lasting seventy-one hours. The patient was a *primipara*. The position was left occipito-posterior. Manual rotation was followed by instrumental delivery. The cord was twice round the neck.

Number 1092.—Labour was long, lasting thirty-six hours. The patient was a *primipara*. The membranes ruptured prematurely. Meconium was passed before the os was fully dilated.

Number 1320.—The patient was a *primipara*. The child weighed nine pounds. The presentation was occipito-anterior. Disproportion was present. The high forceps operation was used. The cord was round the neck.

Number 1872.—The patient was a *primipara*. Labour was long, lasting twenty-five hours. The low forceps operation was used. Presentation was occipito-anterior.

The still-births from unclassified causes were as follows:

Number 908.—The patient was a *primipara* aged twenty-four years. Delivery was natural. No fœtal movements were felt for thirty-five hours before delivery. The first stage lasted five hours and the second stage two hours.

Number 1051.—Twins were born. One was a papyraceous fœtus, the second was still-born.

Number 1512.—The child was large, it weighed ten and a half pounds. The position was occipito-anterior. Delivery was instrumental. At *post mortem* examination no abnormality was discovered.

Number 1533.—Delivery was normal. The mother suffered from pulmonary tuberculosis.

Number 1663.—The patient was a *primipara*. The presentation was occipito-anterior. The low forceps operation was used. No difficulty was experienced in delivery. Death was due to cerebral hæmorrhage. (A *post mortem* examination was performed.) The fœtal heart sounds were not heard before the application of forceps.

Number 1704.—The patient was a *primipara* aged sixteen years. Labour was induced by quinine and castor oil, as the estimated date before its onset had passed. Three doses of quinine were given at intervals of two hours, the dose being ten grains. Labour started immediately after the third dose. Delivery was natural, labour lasting twelve hours. Though there was no evident cause of death, a *post mortem* examination was refused.

Number 1903.—The patient was a *primipara*. Natural delivery occurred after a labour of twenty-one hours. The fœtal heart was not heard at the commencement of labour. The mother suffered from mitral stenosis and had a severe attack of influenza two days before the onset of labour.

Instrumental Deliveries.

Among the 469 instrumental deliveries morbidity was reported on 48 occasions, 10·23%. Only one case of "failed forceps" has been reported, and this was followed by internal version.

Placenta Prævia.

Short reports of the cases of *placenta prævia* are as follows:

Number 53.—The patient had had six children. Repeated hæmorrhages occurred during the last eight weeks of pregnancy. Labour was normal. Mother and child lived.

Number 106.—*Ante partum* hæmorrhage occurred two days before labour started. The edge of the placenta could be felt. As soon as labour started the os was rapidly dilated and delivery expedited. Delivery was followed by a *post partum* hæmorrhage. The mother and child lived.

Number 147.—The patient had had five children. *Ante partum* hæmorrhage occurred one month before the estimated date of the confinement. More severe bleeding

occurred a fortnight after the first. The fœtal heart was not heard. Version was performed. The mother lived, the child died.

Number 494.—The patient had had five children. A diagnosis of central *placenta prævia* was made. The vagina was plugged with gauze. Six hours later the os was dilated, the placenta was pushed aside and forceps were applied. The mother and child both lived.

Number 755.—The patient was a *primipara*. Repeated hæmorrhages occurred between the twenty-eighth and thirty-third weeks. The condition of the mother was becoming serious. It was decided to perform Cæsarean section. The mother lived, but had a stormy post-operative experience.

Number 974.—The patient had had one child. Free *ante partum* hæmorrhage had occurred. A marginal *placenta prævia* was felt. Delivery was natural, the first and second stages lasting eight hours. Delivery was followed by *post partum* hæmorrhage. Mother and child both lived.

Number 1124.—The patient had had one child. Slight hæmorrhage occurred at the commencement of labour. Labour was allowed to continue for seventeen hours and then fairly severe hæmorrhage occurred. The os would admit only one finger. A diagnosis of central *placenta prævia* was made. Cæsarean section was performed. Mother and child both lived.

Number 1215.—The patient had had four children. *Ante partum* hæmorrhage occurred. A diagnosis of marginal *placenta prævia* was made. Internal version was performed. A living child was born, but *post partum* hæmorrhage occurred which resulted in the death of the mother.

Number 1540.—The patient had had three children. Repeated *ante partum* hæmorrhage occurred. A fortnight before the estimated date of the confinement the vagina was packed. Delivery was instrumental. Mother and child both lived.

The patient had had three children. She was thirty-one years of age. The estimated date of the confinement was October 13, 1930. Slight blood-stained vaginal discharge was noted from September 5, 1930, until the time of admission on September 23. Breech presentation was diagnosed and under general anæsthesia external version was performed. A brisk hæmorrhage occurred, and this was so severe that the vagina was tightly plugged with gauze. At midday on the following day labour pains started strongly and the gauze was removed. The head presented and the os was fully dilated. The placenta was presenting alongside the head. Forceps were applied, but delivery failed. The cord prolapsed. No pulsation was found in the cord, so internal version was performed under general anæsthesia, and the child was delivered by the breech, followed immediately by the placenta. The child breathed at once. Mother and child both did well.

Among the nine cases of *placenta prævia* one mother was lost.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association.

Tannahill, Robert William, M.B., 1931 (Univ. Sydney), "Eastwell", Cammeray Avenue, North Sydney.

Warden, Douglas Arthur, M.B., 1930 (Univ. Sydney), "Tabourie", Muston Street, Mosman.

Public Health.

QUEENSLAND.

THE attention of medical practitioners in Queensland is drawn to certain provisions of *The Health Acts Amendment Act of* 1931 which have important bearing on their practice.

The definition of "venereal disease" has been altered and now reads as follows:

> Gonorrhœa, ophthalmia neonatorum, syphilis, soft chancre, ulcerative granuloma, and venereal warts.

The definition of notifiable disease has been repealed and the following definition substituted:

> "Notifiable disease" includes ancylostomiasis (hookworm), acute anterior poliomyelitis, anthrax, Asiatic cholera, bilharziasis, Brill's disease, diphtheria, bacillary dysentery, amœbic dysentery, epidemic cerebrospinal meningitis, encephalitis lethargica, filariasis, leprosy, malaria, plague (bubonic or Oriental), puerperal fever, puerperal pyrexia (and which shall be taken to mean any febrile condition, other than puerperal fever, occurring in a woman within fourteen days after childbirth or miscarriage, in which a temperature of one hundred point four degrees Fahrenheit (thirty-eight degrees Centigrade) or more has been sustained during a period of twenty-four hours or has recurred during that time), relapsing fever, scarlet fever, smallpox, tuberculosis (all forms), typhoid fever (which shall include paratyphoid fevers), typhus fever (all forms), and any disease, whether infectious or not, which the Governor in Council from time to time by notification in the *Gazette* declares to be a notifiable disease under this Act either generally or with respect to any particular place;

Attention is drawn to the inclusion in the list of "puerperal pyrexia".

The sections dealing with the sale and use of dangerous drugs has been altered.

> **5.** Section one hundred and eleven of the Principal Act is repealed and the following new section is inserted, namely:
>
> "*Dangerous Drugs.*
>
> [*111.*] (*1.*) For the purposes of this Act, the following substances shall be deemed to be dangerous drugs, namely:
>
> (*a*) Morphine;
> (*b*) Cocaine;
> (*c*) Ecgonine;
> (*d*) Diamorphine (commonly known as heroin);
> (*e*) Opium;
> (*f*) Opium charcoal;
> (*g*) Also any salt compound or derivative of any of the above substances;
> (*h*) Coca leaves, Indian hemp, and all preparations of which such resins form the base: also any extract or tincture of Indian hemp; and
> (*i*) Such other substances as may for the time being be prescribed from time to time by the Commissioner with the approval of the Governor in Council.
>
> (*2.*) No person other than a person licensed in such behalf under this Act or otherwise authorised in such behalf under this Act shall at any time have in his possession, or upon any premises or place occupied by him, any dangerous drug, and no person shall supply or procure or offer to supply or procure any dangerous drug to or for any person unless licensed under or authorised by this Act.
>
> (*3.*) The burden of proof that any person is licensed or otherwise authorised under this Act shall in every case be on the defendant.
>
> (*4.*) If any person acts in contravention of or fails to comply with any requirement of this section he shall be guilty of an offence against this Act.
>
> Any person guilty of any such offence shall be liable, on conviction for a first offence, to a penalty of not less than fifty pounds nor more than two hundred pounds or to imprisonment, with or without hard labour, for a term not exceeding six months, or to both such penalty and imprisonment, and in the case of a second or subsequent conviction to a penalty of not

less than one hundred pounds nor more than five hundred pounds or to imprisonment, with or without hard labour, for a term not exceeding two years, or to both such penalty and imprisonment, any law to the contrary notwithstanding, and the court, upon conviction of the offender, may, in addition to any punishment, order that the goods in respect of which the offence was committed shall be forfeited to His Majesty.

(5.) Any member of the Police Force may without warrant arrest any person who has committed, or has attempted to commit, or is reasonably suspected by such member of the Police Force of having committed or of having attempted to commit an offence against the provisions of this section, and any member of the Police Force may detain any person found travelling whom such member of the Police Force may reasonably suspect to have in his possession any dangerous drug contrary to the provisions of this section, and such member of the Police Force may, in the presence of a justice of the peace at a police station at which a justice must be present, search such person and may open and search any pack or package, bag, box, swag, or other receptacle carried or conveyed by such person, and also any vehicle, premises, or place in which such person may be, and such member of the Police Force may seize any dangerous drug found in the possession of such person or in any pack, package, bag, box, swag, or other receptacle carried or conveyed by such person or upon any such vehicle, premises, or place, and may forthwith arrest such person without warrant and detain him in custody until he can be brought before the court to be dealt with according to law.

(6.) The Commissioner may from time to time make regulations for carrying out the objects of this section and in particular for regulating the manufacture, sale, distribution of, and other dealings with dangerous drugs, and prescribing penalties not exceeding five hundred pounds for any breach of any such regulation."

A new section dealing with cyanide of potassium and other poisonous cyanides has been added.

6. After section 111 of the Principal Act (previously inserted) the following new section is inserted, namely:

"*Cyanide of Potassium and other Poisonous Cyanides.*

[111A.] (1.) No person other than one licensed in such behalf under this Act, or otherwise authorised in such behalf under this Act, shall at any time have in his possession or upon any premises or place occupied by him any cyanide of potassium or any poisonous compound of cyanogen.

The burden of proof that he is licensed or otherwise authorised under this Act shall in every case be on the defendant.

(2.) If any person acts in contravention of or fails to comply with any requirement of this section he shall be guilty of an offence against this Act.

Any person guilty of any such offence shall be liable on conviction to a penalty of not less than twenty pounds nor more than fifty pounds, or to imprisonment with or without hard labour for a term not exceeding three months, or to both such fine and imprisonment, and in the case of a second or subsequent conviction to a penalty of not less than fifty pounds nor more than one hundred pounds, or to imprisonment with or without hard labour for a term not exceeding six months, or to both such fine and imprisonment, and the police magistrate dealing with the case may in addition to any further punishment order the goods in respect of which the offence was committed to be forfeited to the Crown.

Any member of the Police Force may without warrant arrest any person who has committed or attempted to commit, or is reasonably suspected by such officer of having committed or attempted to commit any offence against this section, and any member of the Police Force may detain any person found travelling whom such officer may reasonably suspect to have in his possession any cyanide of potassium or any poisonous compound of cyanogen contrary to the provisions of this section, and may, in the presence of a justice of the peace at a police station at which a justice must be present, search such person and may open and search any pack or package, swag, or other receptacle carried or conveyed by such person and any vehicle, premises, or place in which he may be, and may seize any cyanide of potassium or poisonous compound of cyanogen found in the possession of such person or upon such vehicle, premises, or place, and may forthwith arrest such person without warrant and detain him in custody until he can be brought before a police magistrate to be dealt with according to law."

Correspondence.

SPINAL ANALGESIA.

SIR: The article by Dr. A. J. Kennedy in your issue of January 9, 1932, based on his experiences in 470 cases of spinal analgesia, was interesting to me on account of its enthusiastic championship of a method, not infrequently discredited, which I still find, after twelve years' experience, reliable and safe.

My experience has been almost entirely confined to producing analgesia in my own urological operations, and I have always administered the analgesic agent myself. None the less, I always have an anæsthetist in attendance, and in private cases I see that he receives the same fee that would be due to him were he giving the anæsthetic himself. I have the most complete faith in my own technique, but, although always ready to teach it to any anæsthetist who wishes to learn, I have come across very few anæsthetists who are willing to undertake the practice of spinal analgesia. The anæsthetists who attend during my operations perform yeoman service, however, by administering certain injections, occasional drinks and, above all, frequent doses of enlivening conversation.

Dr. Kennedy has evidently obtained very satisfactory results in his series, but I would encourage him to take up again the use of ephedrine as a preventive of blood pressure fall, and I shall make that encouragement practical by submitting shortly to the Editor of this journal a paper on the use of this most potent drug in connexion with spinal analgesia. Dr. Kennedy must surely have been employing an inferior preparation of the drug or else injecting it at wrong times or in insufficient doses when he found that it "did not prevent or minimize the fall in blood pressure". I have found, on the contrary, that you can almost "play tunes" on the blood pressure with it.

Except in my very earliest cases, I have used "Stovaine" for spinal analgesia. I employ Barker's original solution, 5% "Stovaine" in 5% glucose, and inject in the sitting position, without any preliminary medication or local anæsthetic injection. If a surgeon has the ordinary common sense to explain things clearly to the patient before the operation, there should be no question of "mental trauma", that oft-quoted bogy. I like "Stovaine", as it is only necessary to inject about one cubic centimetre of solution, and since about the same amount of cerebrospinal fluid escapes just before one fits the syringe on to the needle, there is hardly any disturbance intrathecally.

Dr. Kennedy's eyperiment on the spinal theca of a calf seems to prove that some leakage of fluid takes place when the theca is punctured with a stout needle, but I do not feel quite sure that such leakage, followed by oversecretion of fluid and consequent raised pressure, is the true explanation of spinal headache. In the first year of my work I had about 5% of headaches, some severe, some milder, but ever since initiating a change in technique the headaches have disappeared, and this over a series of several thousand cases. The change in technique referred to is merely special attention to the amount of movement the patient is subjected to during the first three days after operation. Especially important is the lifting from the

table and the carrying back to bed. The patient must not be moved about jerkily or rolled about unduly, and during the first three days he must not be allowed to sit up. A forty-five degrees position, if a Fowler posture is necessary, will not cause any trouble, but, apart from such a requirement, I allow the patient one pillow on the first day, two on the second, and three on the third. The leakage theory seems tempting, especially as the sitting position after operation seems to increase the proportion of headaches, but can we be sure that the assumption of this position brings on or increases leakage from the puncture hole? When I induce low spinal analgesia, which is confined to the sacral nerve area, I have the patient sitting bolt upright for five minutes after the lumbar injection. If the upright position causes increased pressure in the lumbar portion of the theca, then, in the light of Dr. Kennedy's experiment, a very considerable amount of fluid should escape, especially as I use a stout needle. In spite of this, I never have headaches in my patients, not even the slight ones which Dr. Kennedy mentions as occurring occasionally in his series.

So far I have not used "Percaine", as the amount of fluid to be injected is too great for my liking, and, moreover, it is a hypobaric solution as regards the cerebrospinal fluid, a condition which conflicts with the technique I have insisted upon for years. In this technique one postures the patient, lying on his back immediately after the injection, by means of pillows and a pelvic sandbag, so that a vertebral "well" is formed at any desired height.

The analgesic agent, by its greater specific gravity, diffuses along to the most dependent part of this "well", and also a little beyond it, anæsthetizing the nerve roots in the lumbar region as it diffuses. The sacral nerve roots are anæsthetized by the small amount of solution which diffuses caudally just before you change the patient from the sitting to the supine position. The height of the anæsthesia can be regulated at will, and accords with the position of the bottom of the vertebral "well". For confinement of the analgesia to the sacral nerve area, you simply keep the patient sitting up for five minutes and then operate forthwith.

I have mentioned these details, which can all be consulted in papers of mine published in this journal in June, 1921, and January, 1924, since Dr. Kennedy says that he simply turns the patient on his back and "in three or four minutes the anæsthesia is complete". It seems to me that one should use the physical property of a heavier specific gravity to fuller advantage.

I congratulate Dr. Kennedy on his results and hope he will continue to try and popularize a form of local analgesia, which is employed far too infrequently.

Yours, etc.,

R. J. SILVERTON.

Undated.

Obituary.

MERVYN ATHOL STEWART.

DR. MERVYN ATHOL STEWART, whose death was recorded in a recent issue of this journal, was born on June 4, 1895, at Hawthorn, Victoria. He was the son of the late Alexander Stewart, sometime Deputy Town Clerk of Melbourne.

Mervyn Stewart entered Hawthorn College in 1903 and went to Scotch College in 1910. In 1908 he passed the Junior Public Examination and in 1910 the Senior Public Examination. In 1911 he shared an exhibition in physics and chemistry and gained honours in mechanics and mathematics. He was granted a resident scholarship at Ormond College, but did not go into residence. In 1912 he became a student of medicine at the University of Melbourne. During his course he gained exhibitions in physiology and in therapeutics, dietetics and hygiene. He graduated Bachelor of Medicine and Bachelor of Surgery at a special war-time examination in September, 1916, gaining second place in medicine. He was appointed resident medical officer at the Melbourne Hospital in October, 1916, and Registrar in the following year.

In 1918 Stewart enlisted for active service. He was first of all attached to the Caulfield Military Hospital and was under sailing orders when the armistice was signed. During that year his brother, Cedric Alwyn Stewart, was killed in France. Cedric Alwyn Stewart will be remembered for his work on cerebro-spinal fever in collaboration with Dr. N. Hamilton Fairley. The brothers were close friends and were associated at school, at the University of Melbourne Medical School and at the Melbourne Hospital.

In 1919 Mervyn Stewart obtained the degree of Doctor of Medicine at the University of Melbourne, and in 1921 the degree of Master of Surgery. He was Medical Superintendent of the Melbourne Hospital from 1920 to 1922 and was Acting Surgeon to Out-Patients in 1922. He was appointed Demonstrator in Anatomy at the University of Melbourne in 1925 and Honorary Surgeon to Out-Patients at the Children's Hospital in 1926. The appointment as Honorary Surgeon to Out-Patients at the Melbourne Hospital came in November, 1927.

It will be seen that Stewart was well qualified for surgical practice; his colleagues recognized his ability, and his advice was often sought. He commenced practice in Collins Street in 1922. For a while he conducted a general practice in Malvern as well, but from 1928 he devoted his whole time to consulting practice. He was particularly interested in surgery of childhood and in orthopædics. He was a Foundation Fellow of the Royal Australasian College of Surgeons. His other appointments included that of Health Officer for the Melbourne City Council in 1924; while holding this office, he gave health lectures to the public on the subject of venereal disease. He was Lecturer in Anatomy to the Victorian Branch of the Australasian Massage Association. His early death has been a loss to the art and practice of surgery in Victoria, for he spared no effort to make himself proficient and applied himself with zeal to anything which he undertook.

Dr. Geoffrey Penington writes:

Although Mervyn Stewart's professional attainments and versatile ability were widely known and appreciated, and his genial personality endeared him to his associates and patients, it was in his home and with his more intimate friends that the fullness of his lovable nature and the sterling quality of his character were revealed. He radiated good humour and fellowship, and was a delightful companion who always saw the best in everyone. He was a devoted husband and father, and although possessing the natural charm which makes for success in social life, he rather avoided social activities, and one might say that his family and his home were his most absorbing hobby.

An enthusiastic and successful gardener, his garden stands as a monument to his wide and accurate knowledge, thoroughness in application, painstaking care, hard work and love of beauty. To be in his garden and home and realize the atmosphere of peace and loveliness which pervaded them was to have a deeper understanding of him.

He inspired confidence and it was impossible not to place implicit trust in him. His very presence acted as a tonic, his sympathy soothed, and his calm assurance satisfied while it masked a keen sense of responsibility and a nature which could not but worry until all was well with those under his watchful care.

His own fortitude in ill health was great, and his anxiety was not for himself, but for his loved ones.

It is impossible to measure the loss to his family, his profession and the community, and of no man could it be more truly said that to know him was to love him.

Dr. Basil Kilvington writes:

The death of Mervyn Stewart deprives the profession of one of the most likeable and promising of our younger surgeons. I have been intimately associated with him, for he was my resident at the Melbourne and Caulfield Military Hospitals over a period lasting some years. In all this time I never knew him do a mean action, and he was always the same quiet, courteous and lovable personality.

He volunteered for active service abroad, but was kept some time at the base hospital on account of his organizing

abilities, and he received his sailing orders the day of the armistice.

I know he rather felt the fact that he did not leave Australia, but it was not his doing.

Mervyn Stewart always had surgical ambitions, and he was appointed to the staff of the Melbourne Hospital about three years ago. I do not think we realized the capacity behind his unassuming manner till then, but he soon became one of the most successful teachers and promising operators on the honorary staff.

His sudden death came as a shock to most of his friends, as he appeared robust and healthy. A very promising career has been terminated just as it appeared to be coming on to full bloom. To those of us who worked with him at the hospital it is hard to realize we have seen the last of our colleague.

I climb the hill: From end to end
Of all the landscape underneath
I find no place that does not breathe
Some gracious memory of my friend.

These few lines from "In Memoriam" very feelingly express our feeling towards him.

Dr. J. G. Whitaker writes:

Mervyn Stewart has gone and solved the last problem of all. He was very fond of discussing problems, fully and in detail from his Scotch College days onward. He was a typical Scotch College product and one of the very best, full of character and stability, a plain, straight gentleman.

Everything he did was done brilliantly and well, with thoroughness and honesty predominant; at school, university and at the Melbourne during his time as resident and "Super". At the Melbourne he was beloved and respected by all, and here, I think, he was wonderfully successful, his judgement and ability as "Super" being universally admired. Who will ever forget him at tennis there, where, with myself as partner, he challenged all comers; or when he so successfully led the "Tickle Toe" ballet around the residents' room. He had a rich store of humour, and both his audience and himself derived the greatest enjoyment from his description of quaint doings and sayings of the hospital staff. He was then, and has been ever since, distinguished by his habit of speaking the plain truth, and his fearlessness in doing what was right, demonstrating in these things his fundamental strength.

During the war he enlisted as captain in the Australian Army Medical Corps and served twelve months, but to his sorrow was not sent abroad, the war ending meanwhile. He, like most of us at Caulfield, found time hang very heavily, and our superior officer's explosive, "Good God, Stewart, nothing to do" at least kept him and us amused. Whilst here, he received news of his elder brother's death in France, which affected him deeply.

During his hospital life he had fully equipped himself for his life's work of consulting surgeon. He, like everyone else, experienced something of the turbulence of life's stream after leaving the friendly shelter of the hospital's walls, but he stuck it out and conquered, and we honour him for his courage. He attained one of his dearest ambitions when he was appointed out-patient honorary at the Melbourne, and here, I am assured, he gave of his time and talents unstintedly. His connexion with the Children's Hospital, where he will be so much missed, was, as he often said to me, a wonderfully happy experience for him. We of the Children's Hospital who saw so much of him, can speak in the highest terms of his work there and of his ripe judgement and wisdom. We feel his place can never be quite filled, and staff, students and patients will all be the poorer.

Mervyn brought his family to his beautiful home, and here, amidst his family and flowers, were spent undoubtedly his happiest days. He was a great family man and had a wonderfully happy home. He had fought a good fight and had conquered; his future was rich in promise both for the community and himself. His memory as a wonderful man and dear friend will be forever with us.

Dr. W. W. S. Johnston writes:

An attempt to portray the character of one who has been suddenly taken from our midst is fraught with difficulty. In the case of Mervyn Stewart the effort is accompanied by a sense of profound sadness, for his personality abounded in the friendliness and good fellowship which promote the smooth running of human intercourse. In his presence matters took on a more genial aspect; life's troubles somehow seemed a little less and the future assumed a brighter hue.

His outstanding trait was that of "openness"—a simplicity of character that was very lovable. In his early years after graduation, this possibly led some to see less clearly the solid foundation that was so well laid. He would sometimes complain rather whimsically that others regarded him as taking life in an easy and care-free manner. But it was abundantly clear to those who knew him well, that throughout he acted with all the strength of high ideals and honest endeavour. And in later years, as the responsibilities of his work became increasingly heavy, the steadiness of his character showed out all the more, while his kindly good humour became less the central feature and more the outward charm that rounded off his personality.

He accomplished much in his brief life. For two years he acted as Superintendent of the Melbourne Hospital. During that time he carried out his manifold duties with great ability; always eager to further the interests of the institution, to devote himself to the care of patients and, whenever occasion offered, to seize the opportunity of teaching students. After this followed a period of gruelling work in building up a general practice while awaiting with anxiety the possibilities of a hospital staff appointment. Then, by a turn of the wheel, came within a short space of time the attainment of positions on the honorary staff of two hospitals—the Melbourne and the Children's—with the heavy toll on health and strength thereby demanded.

Now the full strength of his character began to display itself. Increasingly, as his place in the profession became more defined, his valuable qualities came more and more to the front. His past experience as Superintendent of the Melbourne Hospital, his well balanced mind, stored with the fruits of a remarkable memory, his tolerance of view, linked with the courage of his convictions, all combined to establish his position and to forecast a life of great achievement.

He did not spare himself, but he had the joy of work done thoroughly and courageously. And in his leisure hours he had for recompense the home on which he lavished so much care, the garden which he brought to a perfection of beauty, and the family which he loved with all the strength of his nature.

All this is cut off in a moment. And we mourn for what is lost to his profession, to humanity, to his loved ones.

Medical Prizes.

THE ALVARENGA PRIZE.

THE College of Physicians of Philadelphia announces that the next award of the Alvarenga Prize, amounting to about three hundred dollars, will be made on July 14, 1932, provided that an essay deemed by the Committee of Award to be worthy of the prize shall have been offered.

An essay intended for competition may be upon any subject in medicine, but must be accompanied by a written assurance from the author that it has not appeared previously in print, either in whole or in part, in any form, and has not been presented elsewhere in competition for a prize. The essay should represent an addition to the knowledge and understanding of the subject, based either upon original or literary research. It must be typewritten, and in English acceptable for publication without necessity for editing by the committee. Any illustrations should be appropriate and correctly annotated with the text. Essays must be received by the Secretary of the College on or before May 1, 1932.

Each essay must be sent without signature, but must be plainly marked with a motto and be accompanied by a sealed envelope having on its outside the motto of the paper and within the name and address of the author.

It is a condition of competition that the successful essay or a copy of it shall remain in possession of the College; and that it may be published by the author with the consent of the College; other essays will be returned upon application within three months of the award.

The Alvarenga Prize for 1931 has been awarded to Dr. Edgar S. J. King, Melbourne, Australia, for his essay entitled: "The Nature of the Stroma of the Ovary".

Post-Graduate Work.

LECTURES IN ADELAIDE.

THE Post-Graduate Committee of the South Australian Branch of the British Medical Association has made arrangements for two lectures to be given by Mr. C. H. Fagge, F.R.C.S., Vice-President of the Royal College of Surgeons of England. The lectures will be given on the evenings of Monday, February 8, and Tuesday, February 9, at 8.30 o'clock, in the Prince of Wales Lecture Theatre at the University of Adelaide. The titles of the lectures will be: (i) "Acute Abdominal Emergencies", (ii) "Fractures".

Tickets, admitting the holders to both lectures, may be obtained from the Joint Honorary Secretaries, Dr. F. N. Le Messurier and Dr. Leonard Lindon, or from Mr. Dobbie, Secretary of the Branch, 206, North Terrace, the fee for the ticket being half a guinea.

Books Received.

CLINICAL NOTES ON DISORDERS OF CHILDHOOD (PRACTITIONER'S AID SERIES), by D. W. Winnicott, M.A., M.R.C.P.; 1931. London: William Heinemann (Medical Books). Foolscap 4to., pp. 224, with charts. Price: 10s. 6d. net.

THE PSYCHOLOGY OF A PRIMITIVE PEOPLE: A STUDY OF THE AUSTRALIAN ABORIGINE, by S. D. Porteus; 1931. London: Edward Arnold. Demy 8vo., pp. 453, with illustrations. Price: 30s. net.

ANNALS OF ROENTGENOLOGY: A SERIES OF MONOGRAPHIC ATLASES, edited by J. T. Case, M.D.; Volume XIII: Gynecological Roentgenology, by Julius Jarcho, M.D., F.A.C.S.; 1931. New York: Paul B. Hoeber. Demy 4to., pp. 596, with five coloured plates and 273 illustrations. Price: $20.00 net.

ASTHMA AND HAY FEVER IN THEORY AND PRACTICE, by A. F. Coca, M.D., M. Walzer, M.D., and A. A. Thommen, M.D.; 1931. London: Baillière, Tindall and Cox. Imperial 8vo., pp. 873, with 97 illustrations. Price: 45s. net.

MATAS BIRTHDAY VOLUME: A COLLECTION OF SURGICAL ESSAYS WRITTEN IN HONOR OF RUDOLPH MATAS; 1931. New York: Paul B. Hoeber. Imperial 8vo., pp. 409, with illustrations. Price: $10.00 net.

Diary for the Month.

FEB. 2.—New South Wales Branch: Organization and Science Committee.
FEB. 3.—Victorian Branch: Branch.
FEB. 5.—Queensland Branch, B.M.A.: Branch.
FEB. 9.—New South Wales Branch: Ethics Committee.
FEB. 12.—Queensland Branch, B.M.A.: Council.
FEB. 16.—New South Wales Branch: Executive and Finance Committee.
FEB. 23.—New South Wales Branch: Medical Politics Committee.
FEB. 24.—Victorian Branch: Council.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser" page xiii.

MATER MISERICORDIÆ CHILDREN'S HOSPITAL, BRISBANE, QUEENSLAND: One House Physician, One House Surgeon.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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